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Fig. 2. Photograph made at operation in Case 5 after resection of a large arteriosclerotic aneurysm of the abdominal aorta and insertion of a bifurcation graft.

Surgical Treatment of Aneurysm of Abdominal Aorta by Resection and Restoration of Continuity with Homograft.—Michael E. De Bakey and Denton A. Cooley.

SURGERY
GYNECOLOGY AND OBSTETRICS

VOLUME 97 SEPTEMBER, 1953 NUMBER 3

SURGICAL TREATMENT OF ANEURYSM OF ABDOMINAL AORTA BY RESECTION AND RESTORATION OF CONTINUITY WITH HOMOGRAFT

MICHAEL E. DE BAKEY, M.D., F.A.C.S., and DENTON A. COOLEY, M.D., F.A.C.S.,
Houston, Texas

ANEURYSMS of the abdominal aorta constitute a serious and difficult problem in terms of both prognosis and therapy. Although there is some variation in survival rate as recorded by different observers, there is general agreement that it is relatively short, ranging from 6 months to 2 years in well over half the patients with only a few patients living more than several years after onset of symptoms or establishment of diagnosis. In general, treatment has not been satisfactory, and while the condition has been a challenging problem to surgeons for a long time, the various methods of surgical attack have, for the most part, not been fruitful. These may be classified into three categories: (1) those designed to promote thrombosis and fibrotic organization by partial, complete, or gradual occlusion or ligation of the aorta, by the introduction of foreign material, or by periarterial fibroblastic reaction; (2) endoaneurysmorrhaphy; and (3) extirpation of the lesion with or without restoration of blood flow. Only the last procedure may be considered promising or satisfactory since it meets the ideal objectives of surgical therapy, namely, extirpation of the diseased part and restoration of normal function.

For various reasons, however, this approach to the problem has not been fully explored and has been successfully employed in only a relatively small number of cases of sacciform aneurysms (1) or those associated with coarctation. Indeed, a review of the literature reveals only one report (that of Dubost and associate) of its successful application in a case of fusiform aneurysm of the abdominal aorta. For these reasons and because of the gratifying results obtained with this method of therapy in 7 patients with fusiform aneurysms of the abdominal aorta, it seems desirable to report our experience with the procedure, which consists essentially in resection of the aneurysm and involved segment of aorta with restoration of normal blood flow by means of an aortic homograft.

TECHNICAL CONSIDERATIONS

Whereas some modifications in technique were made as experience developed and as indicated by the findings in the different cases, the surgical approach employed and the essential steps in the operative procedure were much the same in all cases. The peritoneal cavity is entered through a midline incision extending from a point several centimeters

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Aided by a grant from the Houston District Chapter of the Texas Heart Association.

below the xiphoid process to a point midway between the umbilicus and the pubis (Fig. 1a). The aneurysm may then be readily exposed by retracting the small intestine and its mesentery to the right and by dividing the peritoneum overlying the aneurysm along with the ligament of Treitz to permit mobilization of the duodenum to the right (Fig. 1b). Considerable care must be exercised in performing the latter procedure in order to avoid injury to the duodenum which is often intimately adherent to the aneurysm. By careful blunt and sharp dissection and with further retraction of the root of the mesentery, the aorta immediately above the aneurysm and just below the origin of the renal arteries is exposed and encircled with umbilical tape, thus to provide a safety measure for the control of hemorrhage. In exposing the aorta in this region, careful effort should be made to prevent injury to the overlying left renal vein. The inferior mesenteric artery, which in most instances is thrombosed, is divided between ligatures, to permit further mobilization of the structures along the left lateral wall of the aneurysm (Fig. 1b). The structures along the right lateral wall, including particularly the mesentery of the small intestine, are mobilized laterally exposing the vena cava to which the aneurysm is often intimately adherent. In such instances, it is preferable not to attempt further dissection to separate these structures. Attention is then directed to mobilization of the bifurcation of the aorta and the iliac arteries. The major difficulty involved in this procedure lies in the separation of the posterior wall of these structures from the underlying left iliac vein. Once these vessels are completely freed, they are encircled with umbilical tapes. In some instances, however, owing to the intimate adherence of the iliac arteries to the underlying vein, it may be preferable to delay further separation of these structures until after occluding clamps have been applied to the aorta and iliac arteries.

After freeing the aneurysm, the aorta, and the iliac vessels to this extent, long-handled atraumatic arterial clamps are applied to the aorta above and to the iliac arteries below. Just before occluding the aorta 10 milligrams of heparin solution are injected into the aneurysm to combat the possible thrombophilic effect of the retarded blood flow in the distal vascular bed. The aneurysm is then excised between the clamps. In cases in which the vena cava and left iliac vein are intimately adherent to the posterior wall of the aneurysm, this portion of the aneurysm is not removed. In such cases the ostia of the lumbar arteries presenting in this remaining posterior wall of the aorta are oversewn. In other cases in which the posterior wall of the aneurysm and aorta can be readily separated from the vena cava, these lumbar vessels may be individually ligated.

The previously prepared aortic homograft is then measured to bridge the defect. In cases in which the bifurcation is preserved, it is preferable to employ a homograft of thoracic aorta because it provides better approximation owing to its slightly larger caliber. If the bifurcation must be removed with the aneurysm, the homograft must consist of the terminal abdominal aorta and main iliac vessels (Fig. 2). The proximal anastomosis is performed first. In cases involving a bifurcation graft, it is desirable to permit blood flow through one iliac anastomosis during the performance of the other. This may be done by applying an occluding clamp to the other iliac segment of the graft and removing the clamps on the aorta and the anastomosed iliac artery (Fig. 1c). The method of anastomosis employed consists of a simple continuous through-and-through suture of No. 0000 arterial silk. It is believed that this method of anastomosis provides satisfactory results and facilitates the performance of the procedure in the presence of atheromatous changes in the aortic wall. The operative procedure is completed by approximating the ligament of Treitz and posterior peritoneal margins overlying the aorta and homograft and by closure of the abdominal wound.

All of the homografts used in these cases were obtained at autopsy under sterile conditions from relatively young and healthy individuals dying from injury or hemorrhage. The grafts were preserved at 4 degrees C. in media as recommended by Gross and associates. The period of preservation prior to use varied from 3 days to 25 days. Cultural

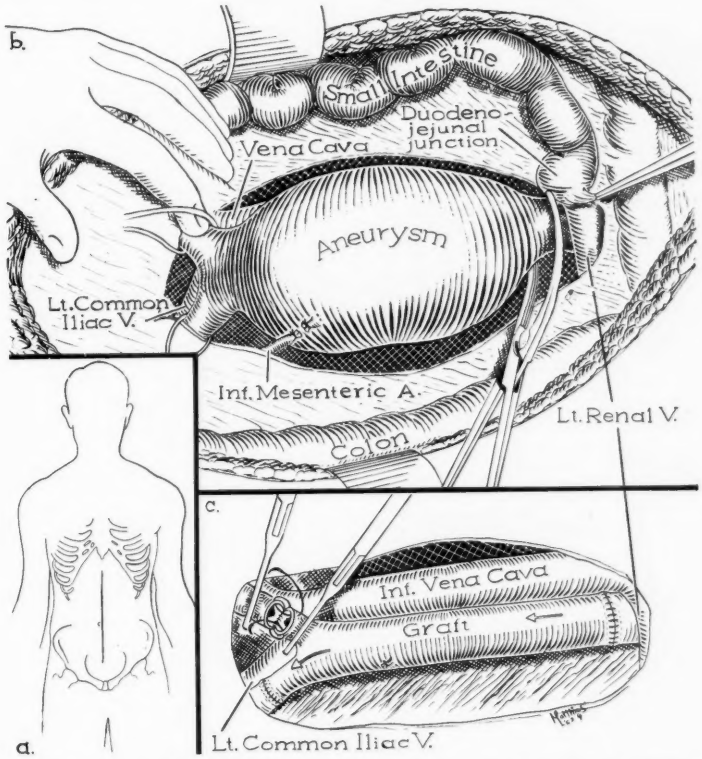

Fig. 1. Drawing showing operative technique of resection of aneurysms of the abdominal aorta with restoration of continuity by aortic graft. a, The abdomen is opened through a midline incision. b, The aneurysm is exposed by incision of the ligament of Treitz and root of the mesentery after displacing the small intestine to the right. c, The defect in the aorta is bridged by a preserved aortic homograft which is sutured in place. In a bifurcation graft of this type after completing the proximal aortic and one of the iliac anastomoses, an occluding clamp is applied to the other iliac segment of the graft and the remaining clamps removed to permit blood flow through the graft during completion of the other anastomosis.

studies of the media revealed no bacterial contamination. In general, these grafts proved satisfactory although the aorta and iliac arteries in most of the patients were of slightly greater caliber than these respective vessels in the grafts. This discrepancy was overcome by beveling the anastomotic ends of the graft to provide a larger circumference. Whereas these preserved homografts proved generally satisfactory, a plastic prosthesis as described by Voorhees has definite advantages.[1]

[1]Use of a freeze-dried aortic homograft recently has impressed us with the advantages of this method of graft preservation.

CLINICAL OBSERVATIONS

As may be observed from the appended brief case reports, the age of our patients ranged from 47 to 74 years, and the aneurysms were considered of arteriosclerotic origin in all but 1 (Case 3), the latter being probably of syphilitic origin (Table I). The significance of these observations lies in the fact that neither age nor arteriosclerosis, per se, constitute contraindications to the successful application of the procedure. Like others, we were formerly of the opinion that the procedure would be more applicable to syphilitic

TABLE I.—DATA ON PATIENTS WITH ANEURYSM OF ABDOMINAL AORTA TREATED BY RESECTION AND RESTORATION OF CONTINUITY BY AORTIC HOMOGRAFT

Case	Age Sex Race	Etiology	B.P.	Date of operation	Period of aortic occlusion, min.	Graft		Results	Remarks
						Age of donor, years	Period of preservation, days		
1. J. J. S.	62 M W	Arteriosclerosis	140/90	11-6-52	57	32	5	Recovered	Lumbar sympathectomy
2. E. J.	71 M W	Arteriosclerosis	140/90	12-4-52	50	24	12	Recovered	Jaundice (cholecystectomy and choledochostomy duodenotomy with removal of common duct stones)
3. J. M.	47 M C	Syphilis	100/80	1-12-53	52	27	25	Recovered	7-12-51: Thoracotomy resection of innominate aneurysm. 12-8-51: Laparotomy cellophane wrapping abdominal aneurysm
4. A. W.	74 F W	Arteriosclerosis	180/100	1-27-53	105	18	13	Died 2-10-53	Progressive uremia followed by secondary hemorrhage
5. G. M.	64 M W	Arteriosclerosis	130/60	2-24-53	48	26	3	Recovered	
6. H. D.	67 M W	Arteriosclerosis	200/100	2-24-53	102	21	10	Recovered	
7. V. C.	60 M W	Arteriosclerosis	170/90	3-16-53	75	26	14	Recovered	8-7-52: Laparotomy cellophane wrapping abdominal aneurysm

lesions in which there is a tendency to produce a firm but relatively supple leathery arterial wall more suitable for suture than that involved by arteriosclerotic changes. Fortunately, this has not proved to be as serious a limiting factor as was formerly believed, the anastomotic procedure having been found technically feasible in all of our cases in spite of rather extensive atheromatous changes in some. This observation assumes additional significance in the light of the recent evidence, shown by Maniglia and Gregory, indicating the diminishing incidence of syphilis and the rapidly increasing frequency of arteriosclerosis as the cause of these aneurysms.

Another important factor of surgical significance lies in the location of these lesions. It may be observed that in all of our cases the aneurysm was situated in the segment of aorta below the origin of the renal arteries. Although in some instances (Cases 1, 3, 5, 7) it involved the bifurcation, it was possible in all cases to occlude the aorta and to secure a sufficient margin of its wall just below the renal vessels to permit resection and anastomosis. This has been a well recognized pathologic feature of these lesions, but it may now be regarded as a fortuitous circumstance that facilitates application of this procedure.

Aortography was employed as a diagnostic procedure in 5 patients. This was done by translumbar aortic puncture with rapid injection of 20 to 25 cubic centimeters of 70 per cent diodrast or urokon solution, and in general provided satisfactory visualization of the aorta and its tributaries (Fig. 3). The usefulness of the procedure lies in its confirmation of the diagnosis and in providing more precise information concerning the extent and location of the aneurysm, particularly in its relation to the renal arteries and the bifurcation (Fig. 3). It is our opinion, however, that while aortography is of value in providing information of this kind, it should not replace exploratory laparotomy in the final determination of feasibility of operation.

In general, the postoperative course of these patients has been relatively benign and satisfactory. Except for the small amount of heparin (10 mgm.) injected into the aneurysm at the time of operation, anticoagulants have not been used and in our opinion are unneces-

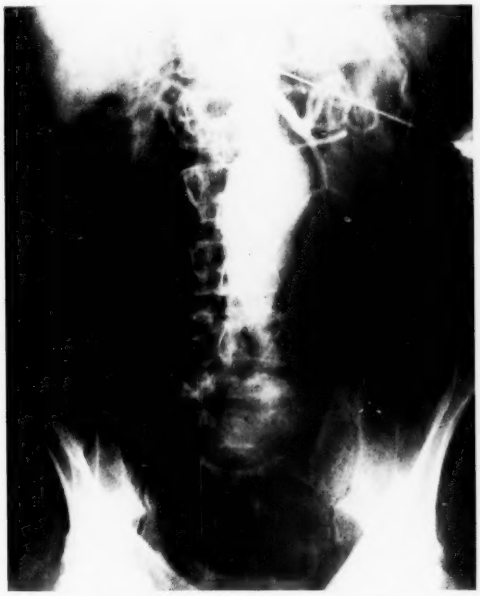

Fig. 3. Aortogram by translumbar puncture in Case 7 showing aneurysm of the abdominal aorta arising approximately 3 centimeters below the renal arteries and involving the bifurcation.

Fig. 4. Aortogram in Case 4 which shows a large partially thrombosed aneurysm of the abdominal aorta. An excessive amount of contrast medium entered the right renal artery producing dense opacification of the kidney.

sary. The period of aortic occlusion varied from 48 to 102 minutes and averaged about 1 hour (Table I), but none of the patients showed any manifestations of arterial thrombosis or residual ischemic changes as a consequence of this period of occlusion. Indeed, circulation in the lower extremities appeared to be improved following operation, and it is doubtful that lumbar sympathectomy, as done only in the first case, is of any value in combatting the possible ischemic effects of aortic occlusion. In this connection, it is of interest to observe that no manifestations of renal or cord damage occurred in 2 of our patients with aneurysms of the lower thoracic aorta after the aorta was occluded in one for 22 minutes (1) and in the other for 45 minutes (2).

The only death in the series (Case 4) occurred on the thirteenth postoperative day following progressive uremia and secondary hemorrhage. Although prior to operation this 74 year old patient showed definite evidence of impaired renal and cardiac function and was thus recognized as a poor surgical risk, operation was recommended because of the rapidly increasing severity of manifestations and apparent imminence of rupture of the aneurysm. The aortogram done immediately prior to operation revealed that a large amount of the injected contrast medium had entered the right kidney (Fig. 4), raising the question of further renal damage which was also indicated by the necropsy findings.

Two of the patients (Cases 3 and 7) in this series had been treated by wrapping the aneurysm with polythene film containing dicetyl phosphate, 13 months and 7 months, respectively, prior to resection. In both instances the wrapping procedure was considered satisfactory as regards sterilization of the material as well as its application to the lesion. Nonetheless, in both cases the aneurysms progressively enlarged, and at the time of resection there was only minimal evidence of fibrosis about the aneurysmal wall. Indeed, in several

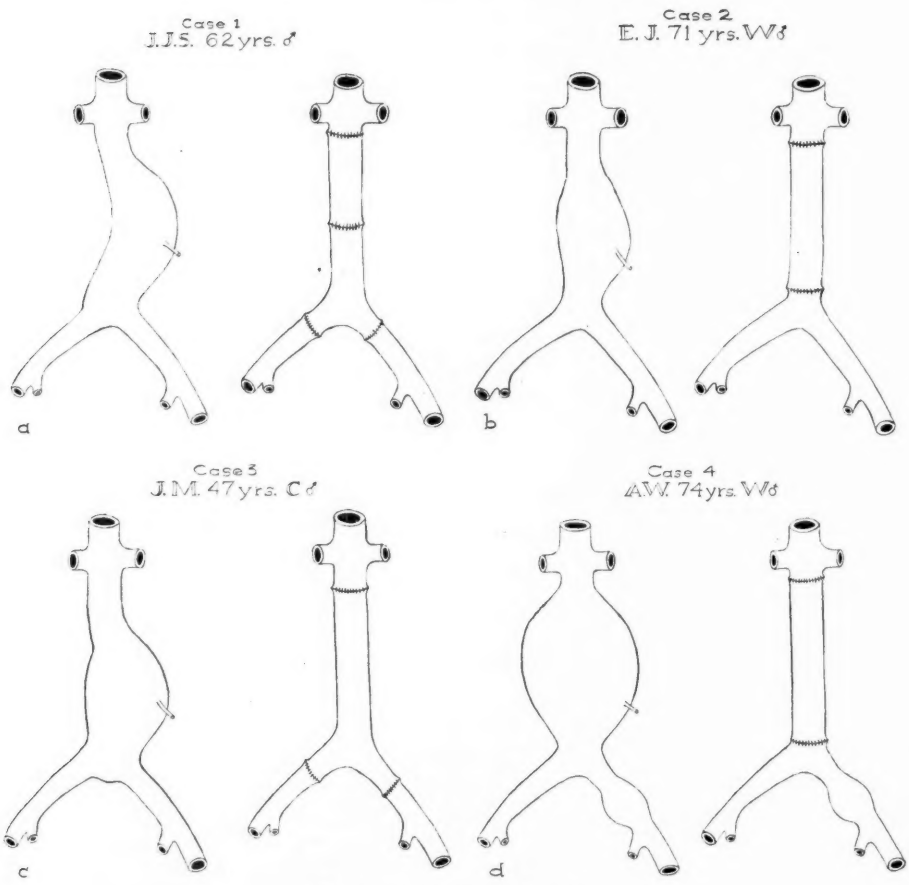

Fig. 5. (*Legend on opposite page.*)

places the wall of the sac seemed much weaker than at the time of the previous operation. These observations as well as other experience with this procedure have raised considerable doubts in our minds regarding the efficacy of the wrapping procedure.

Whereas the immediate results of resection of these aneurysms are considered highly gratifying, it is realized that it is much too early to determine the ultimate results and thus to evaluate properly this method of treatment. As shown by Gross and associates, there is reason to believe, however, from experimental observations on aortic homografts and the long term clinical results obtained by their use in coarctation (4, 5) that it provides a better approach to the problem than any of the previously employed methods. Its ultimate clinical evaluation as well as certain technical considerations concerned with its performance, including the most desirable material for restoration of continuity, must await further observation and experience.

CASE REPORTS

CASE 1. J. S., 62 year old white male, was admitted to Jefferson Davis Hospital on October 16, 1952, complaining of sudden onset of acute low back pain of 2 weeks' duration. The pain was occasion-

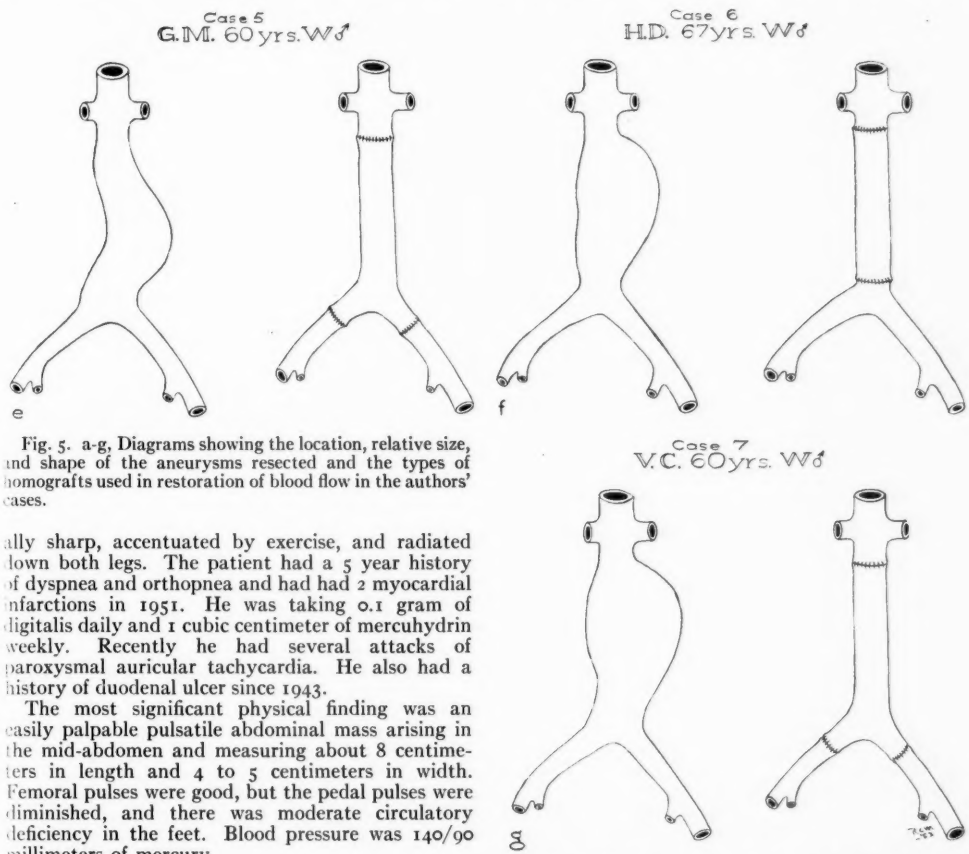

Fig. 5. a-g, Diagrams showing the location, relative size, and shape of the aneurysms resected and the types of homografts used in restoration of blood flow in the authors' cases.

ally sharp, accentuated by exercise, and radiated down both legs. The patient had a 5 year history of dyspnea and orthopnea and had had 2 myocardial infarctions in 1951. He was taking 0.1 gram of digitalis daily and 1 cubic centimeter of mercuhydrin weekly. Recently he had several attacks of paroxysmal auricular tachycardia. He also had a history of duodenal ulcer since 1943.

The most significant physical finding was an easily palpable pulsatile abdominal mass arising in the mid-abdomen and measuring about 8 centimeters in length and 4 to 5 centimeters in width. Femoral pulses were good, but the pedal pulses were diminished, and there was moderate circulatory deficiency in the feet. Blood pressure was 140/90 millimeters of mercury.

Laboratory findings were not significant. Roentgenogram of the abdomen revealed a slightly calcified abdominal aorta and a more heavily calcified aneurysm. Aortogram confirmed the diagnosis of an aneurysm arising 4 centimeters below the renal arteries and involving the bifurcation.

On November 6, 1953, under ether anesthesia the abdomen was explored through a left paramedian incision. There was a healed duodenal ulcer. The fusiform aneurysm was exposed and involved the aorta from a few centimeters below the renal arteries to the bifurcation. After it had been completely mobilized, it was removed between clamps and replaced with an aortic homograft including the bifurcation (Y graft) (Fig. 5a). The period of total occlusion was 57 minutes and of occlusion of the left iliac artery 77 minutes. Pulsations were good in both extremities. After completing the anastomoses the graft functioned well but was too long for the defect it bridged and coiled up anteriorly with each systole. Consequently, 2.5 centimeters were resected from the center of the graft and again end-to-end anastomosis was done. The total occlusion time for this last procedure was 25 minutes. The lumbar sympathetic ganglia were removed bilaterally and appendix was removed. He tolerated the operation well.

On the day following operation, the patient showed some manifestations of deep venous thrombosis in the right leg. On the third postoperative day he coughed violently and had a wound dehiscence requiring secondary closure. The postoperative course thereafter was uneventful, and the patient was dismissed on the thirtieth postoperative day. Circulation in the legs was definitely improved and the patient has remained well.

CASE 2. E. J., 71 year old white male, was admitted to the Methodist Hospital, December 1, 1952, complaining of recurrent episodes of pain in the right subcostal region of the abdomen for about 5 years, becoming progressively worse during the past 3 months. The patient had no previous episodes of jaundice or clay colored stools, but both were

present on admission. There was a net weight loss of 20 pounds. On a previous hospitalization, cholecystogram revealed a nonfunctioning gallbladder.

Physical examination and laboratory studies suggested the diagnosis of obstruction jaundice due to common duct obstruction by gallstones. The blood pressure was 140/90. The heart was not enlarged, and there were no murmurs. There was a readily palpable pulsatile mid-abdominal mass approximately 9 by 6 centimeters in size. The femoral pulses were palpable, but the dorsalis pedis and posterior tibial pulses were absent. An aortogram demonstrated an aneurysm of the abdominal aorta arising below the renal vessels.

On December 4, 1952, under ether anesthesia, a long midline incision was made. A scarred contracted gallbladder was exposed, and a cholecystocholedochal fistula was discovered. Exploration of the common duct along with duodenotomy revealed 6 large and numerous smaller stones which were removed and a T tube was inserted. Duodenal opening was closed and gallbladder removed.

The aneurysm was then exposed at the root of the mesentery by displacing the intestines to the right and descending colon to the left and incising the posterior peritoneum. The inferior mesenteric and 3 pairs of lumbar arteries were ligated and divided. The aorta was occluded proximal and distal to the aneurysm which was removed, the upper clamp being just below the renal arteries and the lower clamp just above the bifurcation of the aorta. The fusiform aneurysm was 9 centimeters long and 6 centimeters in diameter. A straight aortic homograft was then sutured in place with No. 0000 arterial silk and a continuous over-and-over suture (Fig. 5b). When the clamps were released, there was negligible bleeding from the site of the anastomosis. Blood flow was re-established within 50 minutes of the initial clamping. The appendix was removed and the incision was closed.

On the first postoperative day the patient had warm extremities and good pulsations of the dorsalis pedis and posterior tibial arteries. The postoperative course was uncomplicated. The T tube was removed on the eighth postoperative day, and the patient was discharged on the sixteenth postoperative day and has remained well.

Case 3. J. M., 47 year old negro male, was admitted to the Jefferson Davis Hospital, January 8, 1953, because of intermittent episodes of severe pain in the mid-abdomen not related to meals and accompanied by severe vomiting. The symptoms had first appeared in August 1951 and have necessitated periodic hospitalizations.

In July 1951 a massive syphilitic aneurysm of the innominate artery was resected and had shown no evidence of recurrence (Case 3, ref. 1).

Because of the abdominal symptoms he was hospitalized again, and aortogram confirmed the diagnosis of aneurysm of the abdominal aorta arising just below the renal arteries. On December 8, 1951, the aneurysm was explored and wrapped with dicetyl phosphate polythene film. Since that operation the symptoms were not relieved and the aneurysm seemed to be enlarging.

On this admission the blood pressure was 100/80 millimeters of mercury and the heart was moderately enlarged to the left. Pulsations in the abdominal mass were more marked than at the time of wrapping 13 months previously, and the lesion appeared to be larger. Pulses in the lower extremities were good. Significant laboratory findings were limited to a mildly positive serologic test for syphilis (Kolmer and Klein).

On January 12, 1953, under ether anesthesia the abdomen was explored through the previous paramedian incision. It was surprising how little fibroplasia had accumulated about the aneurysm which had almost doubled in size and measured about 8 centimeters in length and 4 centimeters in width. The lesion involved the bifurcation so after excision a Y graft was inserted (Fig. 5c). The anastomosis was done with No. 0000 arterial silk sutures, and the period of aortic occlusion was 52 minutes.

The patient was ambulatory on the second postoperative day and was discharged in good condition on the eighth day. He has remained free of symptoms for 3 months.

Case 4. A. W., 74 year old white female, was admitted to the Methodist Hospital on January 23, 1953, for a painful pulsating mass in the left upper quadrant of the abdomen of about 1 year's duration with increasing symptoms of pain, nausea, and vomiting during the past several weeks. She had some shortness of breath on exertion but no nocturnal dyspnea. The patient had received treatment for hypertension for several years and had experienced nocturia twice nightly and urinary frequency.

Examination revealed a tender pulsating mass in the left upper abdominal quadrant extending down to the umbilicus. The patient had weak dorsalis pedis and posterior tibial pulses bilaterally. Blood pressure was 160/90 millimeters of mercury.

On January 22 an abdominal roentgenogram revealed a partially calcified aneurysm of the abdominal aorta. Excretory pyelography showed no abnormalities of the kidneys. Laboratory studies revealed moderate pyuria and a blood urea nitrogen of 26 milligrams per cent.

On January 27, 1953, under light pentothal anesthesia a translumbar aortogram was done injecting 25 cubic centimeters of 70 per cent urokon rapidly. It was noted that the aneurysm extended from the level of the upper border of the second to the upper border of fifth lumbar vertebrae with the lumen smaller than the total mass, indicating the presence of an intraluminal thrombus. There was dense opacification of the right kidney (Fig. 4).

The patient was transferred to the operating room where under ether anesthesia a midline abdominal incision was made and the aneurysm was exposed through the base of the mesentery reflecting the bowel to the right. The aneurysm extended from the renal arteries to a point 1.5 centimeters above

the bifurcation of the aorta. After the preliminary dissection was completed, the aorta was clamped above and below with atraumatic clamps and the aneurysm was resected.

The resected portion measured 17 centimers in length by 7 by 4.5 centimeters, and a portion of the posterolateral wall of the aneurysm was left in situ on the inferior vena cava to which it was densely adherent. A homograft of thoracic aorta was trimmed to size and sutured in place with No. 0000 black silk over-and-over sutures (Fig. 5d). The clamps were released and a small amount of bleeding was controlled with additional sutures. The appendix was removed and the incision was closed. The procedure lasted almost 5 hours, and the aorta was clamped for 105 minutes.

During the first 24 hours after operation the patient produced only 100 cubic centimers of urine although there was no evidence of shock. Oliguria persisted for the next 48 hours, finally being followed by slight diuresis and adequate urinary flow. Blood urea nitrogen rose steadily, and she developed almost continuous nausea. On the tenth day the blood urea nitrogen was 166 milligrams per cent and the creatinine was 6.7 milligrams per cent.

On the thirteenth postoperative day (February 10, 1953) the patient became stuporous and hypotensive. A mass appeared in the right upper quadrant and she died on that day.

Autopsy revealed a massive retroperitoneal hematoma most marked in the region surrounding the right kidney. The graft and the suture lines were intact, and the bleeding had apparently come from a small perforation of the aorta about 2 centimeters proximal to the upper anastomosis, possibly where the occluding clamp had fractured an atheromatous plaque. The right kidney was smaller than the left and showed evidence of cortical necrosis.

CASE 5. G. M., 64 year old white male, was admitted to the Methodist Hospital on February 13, 1953, complaining of increasing abdominal pulsations associated with epigastric discomfort of 5 years' duration most marked in the past few weeks. He experienced shortness of breath and pain in his legs on walking.

The heart was not enlarged, and there were no murmurs. The blood pressure was 130/60 millimeters of mercury. There were occasional moist rales at the bases of both lungs. There was a visible and slightly tender pulsating mass in the epigastrium about 8 centimeters in diameter. Circulatory deficiency in the feet with weak pedal pulses was evident. Laboratory findings were within normal range. Electrocardiogram revealed an abnormal tracing compatible with coronary artery disease. The patient was discharged from the hospital for slow digitalization at home and returned to the hospital February 22, 1953, in better general condition.

On February 24, 1953, under ether anesthesia an abdominal exploration through a midline incision revealed an aneurysm of the abdominal aorta arising about 2 centimeters below the renal artery and extending to the bifurcation of the aorta. After the lesion was completely mobilized from the surrounding structures, the aorta was occluded above and the common iliac arteries were occluded below. The fusiform aneurysm, which was about 12 centimeters long and 5 centimeters in width, was excised and an aortic Y homograft was inserted (Figs. 2 and 5e). The aortic flow had been interrupted for 48 minutes during the procedure.

The course after operation was satisfactory, and circulation to the legs was much improved. About 1 week after operation he developed a troublesome diarrhea which subsided before hospital discharge on the twentieth postoperative day.

CASE 6. H. D., 67 year old white male, was admitted to Methodist Hospital, February 18, 1953, complaining of increasing abdominal pain of 18 months' duration. Shortly after the onset of pain, an exploratory laparotomy was done on the presumptive diagnosis of a duodenal ulcer, and an aneurysm of the aorta was discovered. During the past 5 years he has been treated for hypertension.

The blood examination revealed in the epigastrium a large, some what tender, pulsating mass measuring about 10 centimeters in length and 8 centimeters in width. There were good femoral and fair pedal pulses. Blood pressure was 190/60 millimeters of mercury. Laboratory findings were not significant.

On February 24, 1953, under ether anesthesia exploratory laparotomy through a midline incision confirmed the diagnosis of a large aneurysm measuring approximately 10 centimeters in length and 9 centimeters in width and involving only the aorta from a point 2 centimeters distal to the renal arteries to a point 1 centimeter above the bifurcation. By mobilizing and occluding the proximal and distal segments of aorta, the aneurysm was excised except for a narrow plaque of the sac which was intimately adherent to the inferior vena cava. The defect in the aorta was bridged with a straight aortic homograft, and the aortic clamps were released after 1 hour and 42 minutes of occlusion (Fig. 5f). After operation his condition was good. He developed troublesome urinary retention and moderate postoperative ileus. These symptoms subsided gradually. At the time of discharge from the hospital on March 16, 1953, the femoral and pedal pulses were excellent, and apart from mild hypertensive headaches his condition was satisfactory.

CASE 7. V. C., 60 year old white male, was admitted to Methodist Hospital, August 4, 1952, having noticed 1 month previously a pulsating mass in his abdomen. The mass had caused practically no symptoms, and the circulation to lower extremities was good. The pulsatile mass was located just above the umbilicus and was slightly movable. Aortogram on August 7, 1952, revealed an aneurysm below the renal arteries and extending to the level of the bifurcation of the aorta (Fig. 3). He was transferred to the operating room where a laparotomy was done. The aneurysm was large, measuring about 12 centimeters in length and 6 centimeters in

diameter, and was attached loosely to the vertebrae posteriorly but was adherent to the inferior vena cava at one point. The aorta above the lesion was encircled loosely with polythene film containing dicetyl phosphate, and the lesion itself was wrapped with this material. The patient recovered satisfactorily from the operation and was discharged from the hospital on August 16, 1952.

He was examined periodically after discharge, and when he was seen on February 26, 1953, he complained of increasing abdominal discomfort for several weeks. The aneurysm appeared to be slightly larger and more tender than previously, and he was readmitted to the hospital on March 14, 1953.

On March 17, 1953, under ether anesthesia the midline abdominal incision was reopened. Dissection in the retroperitoneal tissues revealed an almost granulomatous reaction about the aneurysm, but after the polythene film was removed the wall of the aneurysm itself was glistening and soft. The lesion appeared to have enlarged and now extended to involve the aortic bifurcation. The aorta above and the common iliac arteries below were occluded, and the aneurysm was excised leaving a small plaque of tissue which was adherent to the cava. An aortic Y homograft was inserted to bridge the defect (Fig. 5g). The aorta was occluded for 1 hour and 15 minutes before the left iliac anastomosis was opened to the aortic stream. Twenty minutes later the right iliac anastomosis was opened.

He tolerated the operation well, and the postoperative course was satisfactory. The circulation in the lower extremities was good at the time of discharge on the twelfth postoperative day.

SUMMARY AND CONCLUSIONS

1. Aneurysm of the abdominal aorta is a serious condition, most patients surviving less than a year after onset of symptoms or establishment of diagnosis. Most forms of therapy which have been previously employed are not considered satisfactory. For this reason and because of the grave prognosis, a more aggressive attitude directed toward removal of the diseased tissue is advocated.

2. Seven cases are reported to illustrate the procedure of resection of the aneurysm and restoration of normal function by means of aortic homograft. The aneurysm in all but 1 of the patients was of arteriosclerotic origin and all were located in the segment of the aorta below the origin of the renal arteries, fortunately the most frequent site of aneurysm of the abdominal aorta. The age of the patients ranged from 47 to 74 years.

3. There was 1 death in this series, the patient dying on the thirteenth day after operation of progressive uremia and secondary hemorrhage. All the other patients tolerated the operation well and have shown excellent early results.

4. The aneurysms in 2 of the patients had been wrapped with polythene film containing dicetyl phosphate 13 months and 7 months respectively prior to resection. The gross and histologic findings in both cases cast much doubt on the efficacy of this treatment.

5. Although final evaluation of the procedure must await further observation and experiences, on the basis of the immediately gratifying results obtained in this series, it is believed that resection of the aneurysm and involved segment of aorta with restoration of continuity by means of aortic homograft is the procedure of choice in the treatment of this condition.

ADDENDUM.—Since the preparation of this article 5 additional cases have been performed with no deaths and no complications. In all of these cases the aneurysms were arteriosclerotic in origin and involved the bifurcation necessitating its resection. In 1 case in which the aneurysm involved both common iliac arteries, it was possible to utilize an aortic bifurcation homograft that included the external and internal iliac arteries on both sides, thus restoring normal circulation through these vessels. In 3 cases the aneurysms had been treated by wrapping with polythene film containing dicetyl phosphate, 41 months, 23 months, and 9 months respectively prior to resection, with progression in symptoms and enlargement of the aneurysm.

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THE CLINICAL SIGNIFICANCE OF THE INCREASING RESISTANCE OF ORGANISMS TO THE ANTIBIOTICS

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EVERYONE knows that the antibiotics have changed the clinical course of surgical infections during the last 10 years. Where antibiotics are available one seldom sees the overwhelming invasion of the human body by any of the pathogenic organisms. As far as surgical infections were concerned, the prime offenders 15 years ago were the hemolytic streptococcus and the hemolytic Staphylococcus aureus, against which the human body had very little defense because of its inability to build up any immunity. The gram-negative rods, including the colon bacillus, the typhoid bacillus, Pseudomonas pyocyanea and Proteus vulgaris, were of considerably less importance. The organisms of gas gangrene, while they were locally invasive and spread rapidly throughout the tissues in the area of their introduction, more often killed by their toxin than by invasion of the blood stream. The hemolytic streptococcus and the hemolytic Staphylococcus aureus were formerly capable of spreading rapidly through the blood stream to almost any part of the body, often producing multiple abscesses which in turn became foci for further general distribution. The areas most frequently involved in these metastatic abscesses were the liver, spleen, kidneys, lungs, and in young children the long bones, and at times the brain, the heart, the joints, and other serous cavities. Such infections were frequent sequelae of trivial breaks in the surface epithelium of the body, but were more often the result of deeper wounds, particularly civilian street accidents and the gunshot wounds of war.

These infections were of such major importance to the surgical profession that intensive studies were carried out from the earliest times to find out their cause and also to discover some way of terminating them. While Ehrlich was the prime mover and the chief advocate of research in this field, most of those who followed in his footsteps became discouraged and many including Hata and Neufeld and his associates predicted that no effective antibacterial agent could be found which would not be harmful to the body. It was not until 20 years after Ehrlich's death that Domagk, persisting in his search, finally found an agent which could stop the activity of hemolytic streptococci after their introduction into the human body without serious injury, in most cases, from the drug itself. Thus the sulfonamides demonstrated that they could cut down both the mortality and the morbidity of hemolytic streptococcal infections. But when this drug was applied to staphylococcal infections, it was for the most part inert, and staphylococcal septicemia and pyemia continued to occur even in the face of the sulfonamide therapy of staphylococcal infections.

The successful clinical application of sulfanilamide by Leonard Colebrook and his coworkers soon after the discovery of Domagk, as well as the inexpensiveness of the drug and the uniqueness of its action, soon made it popular and it was not long before it was being used indiscriminately not only for hemolytic streptococcal infections, but for all other infections, as well as for illnesses which were not infections at all.

Penicillin was discovered by Fleming in 1927, but his publications did not appear until 1929 and clinical application of this discovery was not made until 10 years later by Florey and Chain and their associates. The difficulty and the high cost of production held back its development until the failure of the sulfonamides to live up to expectations in the treatment of war wounds and other infections encountered in the Army, rendered urgent the need for some other antibacterial agent of promise. Early observations of successful

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treatment with the use of penicillin, not only of hemolytic streptococcal but of staphylococcal infections, gave rise to the hope of successful clinical treatment of Service-incurred infections due to other organisms. With that expectation the Committee on Medical Research and the War Production Board, by subsidizing the pharmaceutical manufacturers, brought about the rapid and ever-increasing production of penicillin in the first years of World War II. There was, in fact, a reasonable supply available in the hands of the Medical Corps when the Channel crossing brought the Allied Forces to the continent of Europe.

Penicillin, however, soon proved to be no panacea. Although danger of invasive infection both by hemolytic streptococci and hemolytic staphylococci was minimized, penicillin was not able to prevent the local infection of war wounds or severe civilian accidental wounds either because organisms were present which were not susceptible to penicillin or because it was rendered inert at the site of local infection by the production of penicillinase. However, just as fulminating hemolytic streptococcal septicemias disappeared from our hospitals with the advent of sulfanilamide, in like manner the staphylococcal septicemias began to disappear as soon as penicillin became available. It should be recalled to mind that in those early days very serious cases of septicemia, which otherwise would have died, were saved by doses of penicillin as small as 5,000 units every 3 hours for a total of 40,000 units a day.

It was not long, however, before it was obvious to certain observers that staphylococcal infections often required more than this amount before they could be brought under control. In fact, in an early paper on the treatment of infections with penicillin, Lyons stated that he thought that staphylococcal infections with septicemia might need as much as 200,000 units of penicillin a day. It may be that even at that early date the staphylococci were beginning to develop a resistance to penicillin. But in the same manner that the medical profession indiscriminately used sulfonamides in the treatment of infections, just so, as soon as penicillin became generally available, the medical profession turned to it with wishful thinking and applied it to anything and everything that came into their office practice which might remotely be caused by bacteria. It was not long also before patients, hearing of the "miracle drug," demanded that penicillin be used in the treatment of their illnesses. In fact, even when penicillin was very scarce, the greatest pressure was brought to bear upon Chester Keefer, who had charge of the civilian distribution of penicillin, to release it for the treatment of so-called "panic cases." People frequently called upon their Congressmen in Washington to get penicillin for the treatment of some constituent who was said to be dying. But with the indiscriminate use and self-treatment with penicillin the dose was often inadequate and the antibiotic did not reach the organisms in a concentration sufficiently powerful to destroy them. Thus, instead of a bactericidal action, the organisms were just held in check for a time. Then the more resistant forms which were not killed by the antibiotic propagated themselves, thus developing progeny resistant to the antibiotic.

Fleming himself, on his first trip to the United States, where he was greeted everywhere as a great benefactor of humanity, said that he feared that the indiscriminate use of penicillin would develop in the community strains which would not respond to penicillin and that as time went on, it would be ineffective in the treatment of infections caused by these organisms. Fleming's fear was amply justified. It was not long before reports began to appear in the medical journals that the resistance to penicillin of staphylococcal strains which were recovered from human infections was increasing from year to year. This was observed first in England and later in many of the study groups in this country. This fact was reported by Garrod and Barber in England and by Spink, Finland, Reiss, and ourselves (29).

Many attempts have been made to determine the mechanism by which the antibiotics inhibit the growth of or destroy bacteria. It is generally believed that in some way they interfere with one or more of the metabolic processes of the organism, prevent-

ing the utilization and absorption of nutrient materials or the excretion of waste materials. There are so many ways in which the metabolism of bacteria can be interfered with that it is not surprising that the different antibiotics act in different ways. A great deal of work has been done in an attempt to discover the mechanism by which organisms develop a resistance or a tolerance to the antibiotic. It is generally believed that in any given agglomeration of bacteria, whether they are multiplying in the animal body or in a culture medium, all stages of their life cycle are represented. Some have just divided, while others are growing up, and still others are just ready to divide. During this process of growth they may vary in their resistance to any deleterious action in the environment around them. Furthermore, certain individuals throughout the life cycle are inherently or genetically more resistant to these external influences than others. When organisms come in contact with a potent antibiotic, some are inhibited or killed promptly, others slowly, while still others may survive and continue to multiply and in so doing produce progeny as resistant as they themselves.

In the early studies on penicillin it was found that most of the strains of Staphylococcus aureus causing surgical infections were susceptible to the drug but a certain number were resistant. The percentage of resistant strains reported in the literature varied from 5 per cent to 10 to 12 per cent. Why certain strains were susceptible while others were resistant could not be determined. However, it was found that some of the resistant strains had the ability of producing penicillinase, which completely interfered with the bactericidal and bacteriostatic action of penicillin. But that is not the whole story. Penicillinase merely destroys the penicillin. Some staphylococci, either through synthesis of glutamic acid within the cell or by an alternate metabolic pathway not attacked by penicillin, are able to metabolize the essential growth factor, whose elaboration is blocked in the sensitive strains. This phenomenon is being studied intensively in several laboratories.

In like manner the mechanism of resistance to penicillin on the part of staphylococci was carefully studied. Abraham, Callow, and Gilliver were among those studying the problem in England, while in this country Demerec and his co-workers (9) in the field of genetics became interested. Demerec's group (8) came to the conclusion that "resistance is not induced by the action of penicillin on bacteria, but originates through mutation, and that penicillin acts as a selective agent to eliminate nonresistant individuals. The degree of resistance can be increased by exposure to higher concentrations of penicillin, and this increase is interpreted as due to a summation of the effects of several independent genetic factors for resistance which undergo consecutive mutation." They conceived the idea that this takes place by means of several steps each time that the organisms come in contact with penicillin.

When Demerec's group carried out similar studies with streptomycin and certain gram-negative organisms (5), they found that resistance developed rapidly. The organisms often became resistant following only one or two contacts with streptomycin, the steps in this case being very high. They might almost be called jumps. A great many workers have studied this problem and patterns have been worked out for all of the commonly used antibiotics. Streptomycin seems to be in a class by itself, permitting as it does the rapid development of resistance by many of the gram-negative rods, while the other antibiotics in general follow the pattern of penicillin.

Eagle has studied this question with great care, attempting to control all of the factors which are involved in this phenomenon. He has stated that "the development of increased bacterial resistance to antibiotics following exposure to the drugs has been demonstrated with every antibiotic and every bacterial species so far studied. In some instances the resistance of the strain gradually returns toward normal levels on repeated subculture in the absence of the drug. In other cases, however, the resistance remains essentially unchanged after hundreds of generations in antibiotic-free media. Two general mechanisms may be postulated for the development of this heritable change. The resistant organ-

TABLE I.—INCREASE IN RESISTANCE TO PENICILLIN COAGULASE POSITIVE STAPHYLOCOCCUS AUREUS

Year	No. of strains	Per cent resistant
1947 to 1948	286	17.8
1949	228	29.1
1950	269	43.5
1951	293	43.0
1952	290	31.1

Recovered from infections treated in the outpatient department, surgical service, Presbyterian Hospital, New York.
Method: Blood agar plate, filter paper disc.

isms may arise as spontaneous mutations which occur in the course of bacterial multiplication. These rare mutants would be present in the original culture, the drug acting as a selective factor. Most workers incline to this view. . . . An alternative possibility is that increased resistance to antibiotics is not the result of a spontaneous mutation, but develops as a direct result of exposure to the drug and is an adaptive change which persists after its removal. . . . These two hypotheses are not mutually exclusive. Gibson and Gibson have suggested that in the case of Escherichia coli and streptomycin, an initial tenfold increase in resistance may be due to adaptation; while highly resistant organisms may arise as the result of a spontaneous mutation. . . . Three possible mechanisms have been suggested: (a) resistant cells which are present in low frequency as spontaneous mutants in the original culture grow out selectively in the presence of antibiotic; (b) a rare spontaneous mutation toward increased resistance occurs during the period of exposure to the drug and is followed by the selective multiplication of the resistant mutant; and (c) an adaptive change occurs in the presence of the drug as the result of which organisms give rise to progeny uniformly more resistant than the original normal culture."

When penicillin was found to be potent against staphylococci in the early stages of its use, it was thought that the staphylococcal problem, which has always been one of the major problems of surgical infections, would be solved in the same way that the hemolytic streptococcal problem had been almost completely controlled by the sulfonamides, reinforced, and perhaps superseded by the potent action of penicillin on these organisms. But by 1948 Barber and Rozwadowska-Dowzenko reported that the percentage of resistant strains isolated from hospital patients had increased in 2 years from 14 per cent in 1946 to 60 per cent in 1948. In 1952 Birnstingl, Shooter, and Hunt, in England, observed that staphylococcal cultures from outpatients only showed a 16 per cent resistance to penicillin, while over 50 per cent of hospital cultures of this organism were resistant. They explained this by suggesting that the inpatients were subject to cross-infection from one patient to another in the hospital. However, another factor must be considered, namely, that the patients who were in the hospital presumably had more serious infections than those who appeared in the outpatient clinic and included also those who had failed to respond to ambulatory treatment.

Dukes, who has become greatly interested in this problem, has found that over 60 per cent of the strains of staphylococci isolated in his laboratory were resistant to penicillin in 1952 and he stated that the percentage is growing yearly. He believes that "a great deal of this development of resistance can be attributed to inadequate dosage, empirical application, and general misuse of this highly important drug." It was his opinion that the frequent tendency of physicians to prescribe "one shot" treatments of penicillin for nonspecific infections is obviously to be deplored. But he points out "the increasing resistance of bacteria which at one time were universally susceptible to the action of antibiotics is by no means restricted to penicillin nor to staphylococci. Similar trends are apparent for other bacteria and other antibiotics." He has been impressed by the difficulty of the problem in surgical infections due to a mixture of organisms, "particularly in surface lesions and subsequent to extensive surgical procedures involving contaminated or infected tissue." He has found that "treatment with any single antibiotic is usually unrewarding . . . less than 20 per cent of such infections have responded to systemic therapy with any one of the broad spectrum antibiotics."

We have ample evidence, therefore, that the more often and the more indiscriminately

any antibiotic is used, the more readily bacteria of all types develop resistance and their progeny, propagating that resistance, fail to respond to treatment with the antibiotic. Monnier and Schoenbach and about the same time Gocke and Finland observed that with the broad spectrum antibiotics, building up a resistance to one develops a resistance to the other. This is particularly true of aureomycin and terramycin. This is also true of chloromycetin with respect to the gram-negative rods but not with respect to the gram-positive cocci. Later Haight, Wilcox, and Finland showed that "the development of neomycin resistance was regularly associated with a significant resistance to streptomycin," but the reverse did not always occur. They added: "None of the strains which were made resistant to streptomycin and neomycin became more resistant to penicillin, aureomycin, terramycin or chloromycetin, bacitracin or polymyxin B, but some of them became more sensitive to these antibiotics."

Our laboratory has been interested in these studies and has confirmed many of the observations already mentioned. We have been particularly impressed by finding organisms resistant in vitro to antibiotics which have been ineffective in the treatment of the case in hand. In many cases no preliminary cultures had been taken, while in others the organisms had been susceptible at first but had become resistant during the course of treatment. This has been observed frequently following treatment both with penicillin and terramycin.

Table I shows our observations regarding the resistance of staphylococci to penicillin obtained from outpatients from 1947 to 1952. However, instead of showing a steady increase in resistance, as mentioned by some other observers, this table shows that in 1951 the figures were almost identical with 1950, and 1952 showed a slight drop. This covers a period during which the broad spectrum antibiotics as well as bacitracin were used with increasing frequency and it may be that these antibiotics have killed off many of the strains which were resistant to penicillin. It may be also that the use of the broad spectrum antibiotics actually has increased the susceptibility of the surviving organisms to penicillin.

TABLE II.—SENSITIVITIES OF STRAINS OF BACTERIA RECOVERED FROM 253 EAR INFECTIONS, OTOLARYNGOLOGICAL SERVICES, JANUARY 1951 TO AUGUST 1952

Organism	No. strains	Per cent sensitive to:				
		Penicillin	Bacitracin	Streptomycin	Terramycin	Polymyxin B
Coagulase positive micrococci	160	63.1	96.9	97.5	90.	4.4
Coagulase negative micrococci	28	50.	78.6	78.6	28.5	12.3
Beta hemolytic streptococci*	28	78.6	96.4	32.2	89.3	10.7
Nonhemolytic Streptococci	38	52.6	92.1	44.7	92.1	4.7
Coli-aerogenes group	16	—	—	68.8	75.	62.5
Proteus group	38	—	—	65.8	15.8	4.7
Pseudomonas group	60	—	—	50.	36.7	73.3

*Hemolytic streptococci not grouped.
Blood agar plate, filter paper disc method.

Every specimen which comes to our laboratory is cultured both aerobically and anaerobically and is tested on the primary culture plates for susceptibility to 5 different antibiotics—penicillin, bacitracin, streptomycin, terramycin, and neomycin. Table II gives the results of a study of cultures obtained from patients on our ear, nose, and throat service, which is under the direction of Dr. Edmund P. Fowler, Jr., who has given permission for the presentation of this table. These are somewhat different from the results of tests on cultures obtained from our surgical service with regard to both inpatients and outpatients, as shown in Tables III and IV. It is seen that with almost every species of organism, those obtained from the inpatients were nore resistant than those obtained from outpatients. In these tables one sees that bacitracin is often the most potent antibiotic against the gram-positive cocci and the so-called "broad spectrum" antibiotics are variable in their action on the gram-negative rods. They are particularly weak against proteus and pyocyaneus, except for polymyxin B, which seems to be the best for pyocyaneus. Table V gives some interesting recent data with regard to neomycin, which has a broad spectrum and is particularly potent against the

TABLE III.—RESISTANCE OF BACTERIA TO ANTIBIOTICS OCTOBER 1, 1952 TO FEBRUARY 15, 1953, SURGICAL BACTERIOLOGY LABORATORY*

	Coagulase pos. staphylococcus		Coagulase neg. staphylococcus		Hemolytic‡ streptococcus		Nonhemolytic streptococcus	
No. strains	In† 227	Out 299	In 50	Out 23	In 55	Out 27	In 115	Out 39
Per cent resistant to:								
Penicillin	44	26	22	22	36	11	51	33
Bacitracin	9	6	16	22	7	7	10	10
Streptomycin	30	6	30	9	74	30	57	64
Terramycin	38	12	50	35	44	4	29	10
Neomycin	2	1	6	0	51	74	65	69

*Cultures from surgical service only.
†In: Inpatients
Out: Outpatients
‡Hemolytic streptococcus not grouped.
Total number cultures: inpatients, 448, outpatients, 375.

proteus bacillus. Its weakness against hemolytic streptococci is of some interest.

It is obvious from all of the preceding data that no clinician, however experienced, can tell when the organisms of any given infection are going to respond to treatment with any of these antibiotics, unless he calls upon the laboratory for help.

In 1949 the senior author took part in a symposium on antibiotics at the Yale University Medical School and at that time he made a plea for reliance upon the laboratory for determining the sensitivity to the available antibiotics of the causative and associated organisms found in every case of surgical infection. The two other members of that symposium, who advocated the use of aureomycin and chloromycetin respectively, both stated that they thought such laboratory examinations were useless because, they said, they were unreliable and frequently good results were obtained when the organisms seemed to be resistant and vice versa. However, with the passage of time and the general dissemination of knowledge with regard to antibiotics and an understanding of the facts which have been mentioned before, and particularly as a result of the experience of failure to obtain results with the antibiotic empirically selected and administered without laboratory tests, more and more doctors are willing to admit that these tests should be done whenever it is possible to obtain a culture.

If we are now willing to concede that tests for sensitivity should be done whenever possible, one may ask what is the very best method of testing for sensitivity in order that it may have a reliable and dependable clinical significance. It is obvious that this test should be done as quickly, as easily, and as cheaply as possible. There are a number of methods that have been devised, some of them cumbersome and some simple, some of them quick and others slow.

Fleming's first observation on the inhibition of the staphylococcus with a mold was in regard to a staphylococcal culture on an agar plate upon which a colony of Penicillium notatum developed. This produced a substance which diffused out onto the plate and destroyed the staphylococcal culture which had already developed several days before. It was not surprising, therefore, that some of the first tests of sensitivity should be done on an agar plate with the purified antibiotic applied to the culture either before or after its inoculation and incubation. The application of the antibiotic was made in a number of different ways in such a manner that it could diffuse out into the agar medium. If potent, it would then produce a zone of inhibition in which the inoculated culture failed to grow.

The simplest method of applying the antibiotic proved to be on a small disc of filter paper. The credit for this has been given by Heatley to C. G. Pope, who used it first in 1940. In this country credit has been given to Bondi and associates, who described a similar method in 1947.

The tube dilution method is one frequently used and is considered by some to be the most accurate from the point of view of a quantitative test. The antibiotic is diluted down through a series of tubes with twofold dilutions. A constant amount of bacterial culture is added and the endpoint is read after 24 or 48 hours, indicating by the clarity of the culture the concentration of the antibiotic preventing the growth of the organism. This is often checked by plating selected tubes at and

beyond the endpoint to see if any organisms grow out in subculture, thus indicating also whether the antibiotic has been bactericidal or simply bacteriostatic.

A turbidimetric method by which the degree of growth of the organism can be measured electrically by the interference of the transmission of light, as compared with the control without the antibiotic, gives some indication of partial as well as complete inhibition.

With the plate dilution method, a series of plates is prepared in which diminishing twofold dilutions of the antibiotic are incorporated in agar, upon which the culture is plated. Generally 5 to 10 per cent of blood is added to the agar before it is plated and after it has solidified, the plate can be divided up into sectors and a number of cultures can be planted in these subdivisions.

Jackson and Finland have demonstrated that when these tests of sensitivity are run concurrently by means of these different methods, inconsistencies often develop. This is particularly true with aureomycin, probably due to the fact that it deteriorates rapidly when in solution and would deteriorate to a varying degree in the different culture media employed for these tests.

Most of the investigators and laboratory workers who have tried to standardize these tests in order to make them of clinical significance, have come to the medicated disc method because of its simplicity, rapidity, and consistency. By this method either the specimen of exudate itself or a small amount of broth in which the specimen has been suspended is smeared evenly over the surface of a blood agar plate. Discs saturated with an antibiotic are placed on the surface of the agar after inoculation. In this way at least 4 or 5, and in some cases as many as 9 different antibiotics may be tested against the culture. After an overnight incubation some indication of the sensitivity of the organism can be clearly shown, providing that there is sufficient growth to cover the plate. The zone of inhibition may be narrow or wide. Although some attempts have been made to quantitate these tests, it is generally admitted by those who use them frequently that this should not be done. However, the results can be classified as indicating resistance, susceptibility, or slight susceptibility.

TABLE IV.—RESISTANCE OF BACTERIA TO ANTIBIOTICS OCTOBER 1, 1952 TO FEBRUARY 15, 1953, SURGICAL BACTERIOLOGY LABORATORY*

	Coliform bacteria		Proteus		Pyocyaneus	
No. strains	In† 171	Out 39	In 81	Out 23	In 35	Out 11
Per cent resistant to:						
Streptomycin	33	13	63	35	77	36
Terramycin	32	18	94	65	77	82
Neomycin	4	0	7	0	73	69

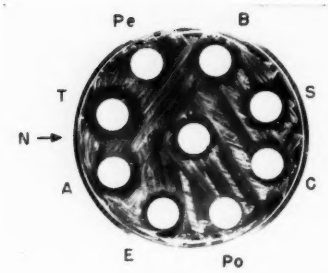
*Cultures from surgical service only.
†In: Inpatients
Out: Outpatients

TABLE V.—NEOMYCIN SENSITIVITIES OF ORGANISMS RECOVERED FROM SURGICAL INFECTIONS APRIL 1952 TO SEPTEMBER 1952

Organisms	No. strains	Per cent sensitive
Coagulase positive micrococci	290	100.0
Beta hemolytic streptococci*	32	21.87
Nonhemolytic streptococci	29	20.62
Coli-aerogenes group	84	91.78
Proteus	87	93.08
Pyocyaneus	37	27.28

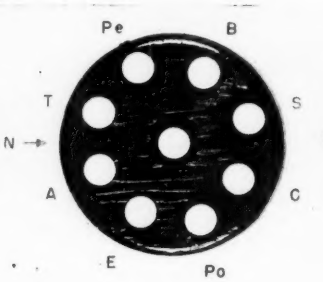
*Hemolytic streptococci not grouped.
Blood agar plate, filter paper disc method.

Bondi, Spaulding, Smith, and Dietz outlined the procedure in 1947, when they tested both penicillin and streptomycin. They used a concentration of 15 units per cubic centimeter of penicillin and 500 micrograms of streptomycin. These concentrations were selected after preliminary trials with various strengths of each one, and they believed that they corresponded to the clinical concentrations obtained by the dose generally used for these two antibiotics. A zone of inhibition more than 20 millimeters, including the radius of the disc, indicated that the organism was very susceptible. If the zone was 10 to 20 millimeters, it was moderately susceptible, and if the zone was less than 10 millimeters, the organism was resistant. With regard to streptomycin, the zone with the very susceptible strains was greater than 15 millimeters



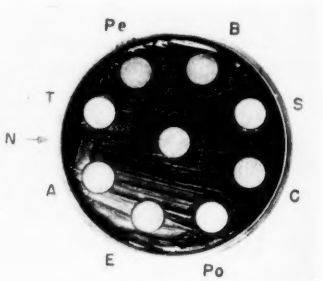
Staphylococcus

Fig. 1.



Proteus

Fig. 2.



Pyocyaneus

Fig. 3.

Fig. 1. Culture of Staphylococcus aureus showing susceptibility to bacitracin, streptomycin, terramycin, aureomycin, and neomycin; slight susceptibility to erythromycin and chloromycetin; and resistance to penicillin and polymyxin B.

Fig. 2. Culture of proteus bacillus showing susceptibility to streptomycin and neomycin; slight susceptibility to polymyxin B; but resistance to penicillin, bacitracin, terramycin, aureomycin, chloromycetin, and erythromycin.

Fig. 3. Culture of pyocyaneus showing susceptibility to polymyxin B but resistance to penicillin, bacitracin, streptomycin, terramycin, neomycin, aureomycin, chloromycetin, and erythromycin.

while those producing a 10 to 15 millimeter zone were called moderately susceptible and those less than 10 millimeters were called resistant. These figures hold only for the thickness of Bondi's plates.

Spaulding and Anderson reported again on this method with regard to the antibiotics later available. They modified the original technique and used concentrations as follows: aureomycin 500 micrograms, bacitracin 75 units, chloramphenicol 700 micrograms, dihydrostreptomycin 500 micrograms, neomycin 300 units, penicillin 50 units, terramycin 500 micrograms. As a routine procedure, however, they limited sensitivity tests on the primary culture to penicillin and aureomycin. If these tests revealed pyogenic species resistant to both of these, representative colonies were subcultured to another blood agar plate on which discs containing antibiotics not previously tested were placed. The concentrations of the antibiotics were selected after rather extensive comparison with the results obtained by tube dilution and agar plate tests and after a trial period of observation for clinical correlation.

The medicated disc method is approved also by Scott and by Gould and Bowie, who point out that for practical purposes it is more important to determine sensitivity than to identify the species. They believe that a dependable report should be possible within 24 hours. However, they stress the importance of great care in preparing the antibiotic dilutions in order that the concentrations may be as accurate as possible. They generally sterilize 100 discs in a bottle and add 1 cubic centimeter, estimating that each disc, therefore, takes up 0.01 cubic centimeter. The solutions are made up 100 times the quantity required for each disc and according to their estimate each disc contains 1 unit of penicillin, 10 micrograms of streptomycin or terramycin, 25 micrograms of chloromycetin, or 50 micrograms of aueromycin.

Eisenberg and Wagner also believe that they have standardized this procedure so that it has real clinical significance, but they believe it should be used solely as a qualitative guide. The concentrations of their antibiotics are based upon the blood levels which can be attained by the usual daily dose of the antibiotic in question, namely, 300,000 units of procaine penicillin, 5 grams of streptomycin twice a day, 0.5 gram of chloromycetin, aureomycin, or terramycin every 6 hours.

Fairbrother and Martyn, in England, use a much higher concentration, namely, penicillin 1,000 units, streptomycin 10,000 micrograms, aureomycin 2,500 micrograms, chloromycetin

2,500 micrograms per cubic centimeter in a strong solution. They estimate that each disc takes up 0.02 cubic centimeter. In other words, each antibiotic is diluted 50 times.

Our own laboratory has used the medicated disc technique since 1945 with great satisfaction. We use filter paper discs, ½ inch diameter, which absorb 0.1 cubic centimeter. The concentration of the antibiotics used is based not only upon the concentration which can be attained in the blood from the usual daily dose, but also takes into consideration the fact that the different antibiotics vary in their diffusibility into the agar from the disc. The concentrations which we have found by long experience to correspond to the clinical results obtained are: penicillin 10 units, bacitracin 40 units, terramycin 100 micrograms, while neomycin and streptomycin have 250 micrograms per cubic centimeter. The results obtained with these concentrations in vitro correspond very accurately with clinical results obtained by the usual upper safe limit of dosage of each of these antibiotics (see Figs. 1, 2, and 3).

If the culture is susceptible to only one of these drugs, that drug is indicated for the treatment of the case. If it is susceptible to two or more, one of them may be selected or the two or more may be combined in the treatment. If there is a mixture of organisms in the culture, as is so often found to be the case with surgical infections, two antibiotics are often needed. If the culture is resistant to all of the tested antibiotics on the primary test, secondary tests are carried out with aureomycin, chloromycetin, polymyxin, and erythromycin. If the micro-organism is resistant on the plate to all of these antibiotics, the inhibiting concentrations for each may be determined by dilutions ranging from 100 to 250 units or micrograms downward in fluid media. Frequently a synergism between two or more of the antibiotics may be demonstrated, whereby fractions such as 1/5 or 1/10 of the minimal inhibiting concentration, when combined, will inhibit the growth of the organism. This is frequently the case when penicillin, bacitracin, or streptomycin are combined, but it must be remembered that aureomycin, chloromycetin, or terramycin may be antagonistic to the other three antibiotics.

For laboratories in small hospitals where these tests are infrequent, one of the several commercially prepared sets of discs may be used. In larger hospitals, however, it is more economical and satisfactory to make up the antibiotic dilutions.

In 1951 the senior author sent around a questionnaire to 196 hospitals in the New York metropolitan area to find out which ones had bacteriologic laboratories where tests could be done, and to inquire whether the surgeons frequently called upon the laboratory for that service. At that time it was revealed that very few such tests were done and few were requested (28). By carrying on a campaign for a more intelligent use of the antibiotics, it seems reasonable to believe that this situation has greatly improved, but we are certain that it still leaves much to be desired. May we make another plea, therefore, for a constant and complete use of the facilities of every hospital bacteriologic laboratory by every surgeon, so that the patients will derive the greatest benefit from our knowledge of these life-saving measures? Only in that way can many infections be brought under control quickly and the patient be preserved from prolonged disability and hospitalization and often be saved from economic disaster. By this procedure also, hospitals can often be spared the heavy expense of free care to patients whose financial resources have been exhausted.

SUMMARY AND CONCLUSIONS

We have briefly presented our own experience and that of others, which clearly indicates that organisms are becoming increasingly resistant to the antibiotics. From year to year more and more cases are failing to respond to antibiotics indiscriminately selected and administered in the treatment of infections.

These facts point to the necessity for determining as quickly as possible by means of laboratory tests the causative organisms and their sensitivity to the available antibiotics. When this knowledge has been gained, the most potent antibiotic or combination of antibiotics should then be employed in the treatment of the case.

Simple and rapid methods of determining bacterial sensitivity to all available antibiotics have been presented. These qualitative tests are significant and indicate the antibiotics most likely to be successful in the control of the infection.

Only in this way can patients receive the full benefit from our present knowledge of these important life-saving measures.

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HISTOLOGIC STUDY OF THE PELVIC CONNECTIVE TISSUE

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THE QUESTION of the supportive function of the pelvic connective tissue has been the subject of discussion for a long time. Likewise, the question of the supportive role of the muscular pelvic floor has received considerable attention in medical literature. There have been, however, wide differences of opinion regarding the relative importance of these two anatomic structures with regard to their biologic significance, physiologic capacity, and anatomic characteristics.

For a long time the view most frequently advanced was that the condensations of the pelvic connective tissue are responsible for the maintenance of the pelvic organs in their normal position. The condensations in the base of the broad ligaments were first referred to by Savage; they were later described by Kocks as cardinal ligaments, and by Mackenrodt as ligamentum transversale colli. Since then, increasing interest has focused upon these structures. The ligaments were described as thick bandlike structures running from the pelvic fascia into the cervix. Their continuation from the pelvic fascia into the vagina was named paracolpos by Fothergill. His view was that "uterus, vagina, and bladder are mainly kept in their place by the lateral combination of unstriped muscle and connective tissue derived from the pelvic fascia." Others looked upon the cardinal ligaments as fibromuscular condensations of the endopelvic fascia.

Other bandlike thickenings of the pelvic connective tissue have been designated as the sacrouterine ligaments and the pubovesicouterine ligaments. The latter have been described as running from the attachment of the ligamentum transversale colli to the cervix, along the side wall of the bladder to the posterior surface of the symphysis. Still other condensations were found as visceral fasciae forming sheaths around the pelvic organs and as the vesicovaginal and rectovaginal septa whose function was to serve as support for the bladder and the rectum. All of these pelvic connective tissue thickenings were said to be attached by means of the cardinal ligament to the pelvic fascia and the pelvic wall. Therefore the cardinal ligament was looked upon as the pivotal supporting structure which holds up the pelvic viscera.

The generally accepted opinion current in anatomic and gynecologic literature is that these radiating masses of fibrocellular tissue form very definite structures and play an important part in maintaining the position of the uterus.

In their extensive research of this question, Curtis, Anson, Ashley, and Jones pointed out the abundance of blood vessels in the fascial sheaths of the viscera as well as in the ligamentous thickenings which support the uterus and the urinary bladder. Curtis and associates had "become increasingly aware of the part played by the blood vessels in the support of the pelvic viscera particularly the uterus and vagina."

Koster stated that there is no bandlike tissue to be found in the base of the broad ligament as described under the name of Mackenrodt ligament.

It has been generally stressed that the condensations of the pelvic connective tissue due to their fixation to the pelvic fascia have the function of suspending the pelvic viscera. According to descriptions in anatomic textbooks, pelvic fascia is the collective name for the fascial layers of the pelvic wall muscles. It consists of the layer covering the internal surface of the obturator internus muscle and the upper surface of the levator ani and coccygeus muscles, and ends posteriorly at the anterior margin of the great sacrosciatic notch.

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The endopelvic fascia fills the pelvic subperitoneal cavity and has been described as originating from the arcus tendineus of the pelvic fascia. The arcus tendineus or the white line of the pelvic fascia is an arcuate thickening of the obturator fascia extending from the ischial spine anteriorly to about 2 centimeters lateral to the symphysis. Tandler described the endopelvic fascia as an extension of the retroperitoneal connective tissue layer of the posterior abdominal wall. He named the entire structure tela urogenitalis. Halban described the endopelvic fascia as the pelvic continuation of the fascia endo-abdominalis which extends over the abdominal walls and thoracic diaphragm and is situated between the parietal peritoneum and the fascial coverings of the abdominal muscles. It is generally accepted that this endopelvic fascia forms fascial sheaths for the pelvic organs, and the names vesical, vaginal, cervical, and rectal fascia serve to designate their specific locations. Also, the vesicovaginal and rectovaginal septa, as well as the pubovesical, vesicouterine ligaments, and the transverse ligament of Mackenrodt have been looked upon as condensations of the endopelvic fascia.

Considerable attention has been paid to these structures because of the supposedly important physiologic function ascribed to them.

This description of certain structural components of the endopelvic fascia was generally accepted until the basic contribution of Byron H. Goff which was made independently of, and corresponded with, the findings of William Blair Bell. Goff's histologic examinations of the tissues between the walls of the pelvic viscera showed that in the normal nullipara there is no tissue in the walls of the vagina, bladder, and rectum which forms well-defined sheaths around these organs and which can be termed fasciae. The anterior vaginal wall and the bladder wall "are loosely held together by a layer of areolar connective tissue." There is a similar areolar connective tissue between the posterior vaginal wall and the wall of the rectum. There is no fascial sheath present other than loose areolar connective tissue. Koster confirmed the findings of Goff, as later did Ricci, Lisa, Thom, and Kron who, in a series of specimens, studied the relationship of the vagina to the adjacent organs from the point of view of reconstructive surgery. Ricci and associates stressed the absence of blood vessels in this loose areolar zone between the anterior vaginal wall and the bladder wall, as well as between the posterior vaginal wall and the rectum. The practical importance of this avascular line of loose attachment had been emphasized previously by Bissel and Rawls in their operative approach to the treatment of cystocele.

The tissue components of the sacrouterine ligaments, which are grossly often not discernible on either side, have recently been studied by Campbell. He found that the matrix of the intermediate and posterior or sacral thirds of the sacrouterine ligaments is composed mainly of loose connective tissue, and he concludes that "they should not be credited with undue supportive value." It can be said that the round ligaments are of no more value as supportive structures than the sacrouterine ligaments. Mackenrodt, at laparotomies, resected a piece of both round ligaments near their origin from the uterus; he then brought the uterus in retroversion and filled the vesicouterine space with intestines. As early as the time when the patient was discharged from the hospital, the uterus could be found in its original anteversion.

In view of the wide divergence of opinion regarding the supportive structures of the pelvic organs, it was felt that a new method of investigation was justified for clarifying our knowledge of the anatomic and functional properties of both the connective tissue structures and the muscular pelvic floor.

The structure of the muscular pelvic floor as found in the living female differs in many essential points from the descriptions in anatomic and gynecologic literature which are based mainly on findings in the cadaver. It is not to be denied that these findings have greatly influenced the analysis of the functional relation of the muscular pelvic floor to the pelvic organs.

We had gained the impression from gross dissections of anatomic specimens that certain structures within the pelvic connective tissue

were probably artefacts. Furthermore, we have found that the histologic examinations hitherto reported were concerned only with limited areas of the pelvic connective tissue. The tissue components of the so-called ligamentous condensations of the pelvic connective tissue were not examined histologically, apparently because the authority of the descriptions of Mackenrodt, Kocks, and Fothergill was generally accepted.

In order to elucidate the question of the relationship of the pelvic organs to the muscular pelvic floor and the connective tissue, we resorted to two methods of examination. For studying the muscular pelvic floor, the new method of x-ray visualization of the pelvic diaphragm in the living female was employed, and our findings will be reported in a later issue of this journal. For studying the connective tissue, microscopic sections were made embracing the whole pelvis in which the blood vessels had been previously injected. This was intended to avoid artefacts in the pelvic connective tissue and to study the tissue components of the ligamentous condensations, the importance of which has been so much stressed for the support of the pelvic organs.

By these combined methods of examination of the pelvic connective tissue as well as of the muscular pelvic diaphragm, it was hoped to clear up moot points and perhaps consolidate the present knowledge of the supportive structures of the pelvic organs.

For the microscopic investigations, 2 adult pelves were used. The first was the entire pelvis of a 23 year old nullipara, removed from the body 3 days postmortem. The blood vessels were first washed out by infusion with physiologic saline solution, the cannula having been inserted into the abdominal aorta. Next, through cannulas inserted into the aorta and inferior vena cava, the arterial and venous portions of the pelvic vascular system were injected with revertex containing 60 per cent latex. Latex was chosen because that substance is dissolved in ether, the solution used in the process of preparation of tissue for histologic sections, leaving the lumina of the blood vessels uncollapsed and open. The entire pelvis was then fixed in a 2 per cent formaldehyde solution. The fixed pelvis was then decalcified in a 5 per cent hydrochloric acid solution. In due course the decalcified pelvis was bisected in the sagittal plane of the body. Each half of the pelvis was immersed in a 1 per cent formaldehyde solution within a metal container and was carefully marked for the exact location of the sections to be made. By placing dry ice around the metal container, the formaldehyde solution with the immersed pelvis became frozen solid. The resulting block of ice was then removed from its metal container, and cut with a highspeed electric saw along the previously indicated markers. One half of the pelvis was cut into parallel sagittal sections; the other half was used for coronal sections. The sections, 1 to 1½ centimeters thick, were subjected to the routine histologic processes. They were finally stained with: (1) eosin and hematoxylin; (2) Mallory for connective tissue; (3) van Gieson for connective tissue; (4) Verhoeff for elastic tissue.

The second anatomic specimen of a pelvis was obtained from the dissecting room of the Department of Anatomy of New York University, where it attracted attention because it was found that the veins of the pelvis were completely filled with coagulated blood, apparently due to antemortem stasis. This pelvis was from a nullipara of unknown age. The blood vessels of this specimen were not washed out with saline solution because it served admirably for comparison with the latex-filled blood vessels of the first pelvis. The histologic sections of this pelvis were made in the same manner as already described.

EXAMINATION OF MICROSCOPIC SECTIONS

Figure 1 shows a paramedian sagittal section through pelvis I. The section passes through uterine body, cervix, and cardinal ligament close to the cervix wall. (The term "cardinal ligament" as generally understood has been retained for purposes of description).

Figure 1a is a highpower photomicrograph of the same section through the cardinal ligament close to the cervix wall as outlined in rectangle *A* (Fig. 1). It is seen that the cardinal ligament consists mostly of blood vessels and loose connective tissue.

Fig 1b is a highpower photomicrograph of a section through the bladder wall embraced in

Fig. 1. *Cl*, cardinal ligament; *R*, rectum; *Sa*, sacrum; *Sy*, symphysis; *U*, uterus, *UB*, urinary bladder; *V*, vagina.

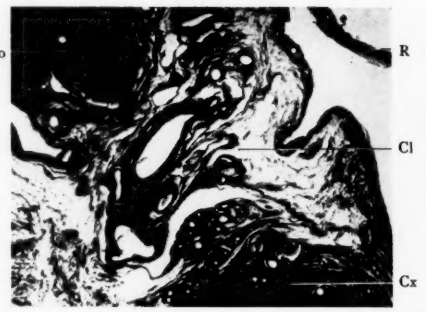


Fig. 1a. *Cl*, cardinal ligament; *Co*, corpus uteri; *Cx*, cervix uteri; *R*, rectum.

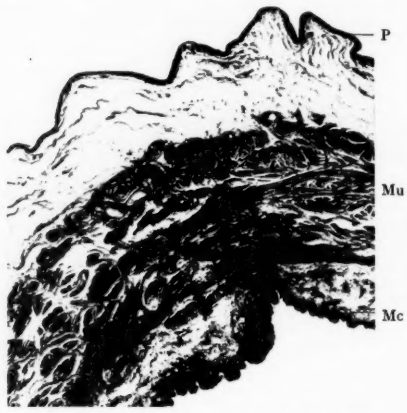


Fig. 1b. *Mc*, mucosa; *Mu*, muscularis; *P*, peritoneum.

rectangle *B* (Fig. 1). Examination of this section reveals that the space between the peritoneum and the muscularis of the bladder is filled with loose areolar connective tissue.

Figure 2 is a saggital section through pelvis I, 0.5 centimeter laterad to the previous section. Here a longer area of the cardinal ligament is seen, and its continuation with the perivesical connective tissue is shown.

Figure 2a is a highpower photomicrograph of the area outlined in rectangle *A* (Fig. 2). It shows that the cardinal ligament consists of loose connective tissue and abundant blood vessels.

It is to be noted that in the sections shown in Figures 1a and 2a which pass close to the cervix wall, no bandlike condensations of connective tissue are present.

Figure 2b represents a highpower photomicrograph of section through bladder wall as outlined in rectangle *B* (Fig. 2). It reveals the presence of loose perivesical connective tissue.

Figure 2c is a highpower photomicrograph of sagittal section through the vaginal fornix and the adjacent part of the bladder wall as outlined in rectangle *C* (Fig. 2). A thin layer of loose areolar connective tissue separates the anterior vaginal wall from the wall of the bladder. Within this tissue layer there is no condensation discernible which could be designated as vesical or vaginal fascia. The continuation of this vesicovaginal layer with the perivesical and parametrial loose connective tissue can be traced in the photomicrograph of Figure 2.

Figure 3 is a sagittal section through pelvis I, about 1 centimeter laterad to the previous section. The section passes outside of the uterus and shows the cardinal ligament in relation to the lateral fornix of the vagina.

Figure 3a is a high power photomicrograph of the area outlined in rectangle *A* (Fig. 3). It shows the blood vessels of the cardinal ligament and its connective tissue component, the latter continuous with the perivesical layer.

Figure 3b is a high power photomicrograph of sagittal section through the intermediate third of the vagina and the adjacent parts of

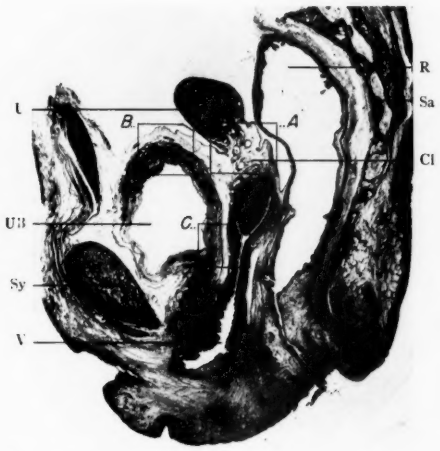


Fig. 2. *Cl*, cardinal ligament; *R*, rectum; *Sa*, sacrum; *Sy*, symphysis; *U*, uterus; *UB*, urinary bladder; *V*, vagina.

the rectum and bladder outlined in rectangle *B* (Fig. 3). It shows the vagina separated from the bladder and rectum by a thin layer of loose connective tissue.

Figure 4 is a section through vagina, bladder, and rectum of pelvis II at approximately the same sagittal plane as Figure 3 of pelvis I. In this specimen the veins are shown filled with blood, as they were ante mortem. The blood vessels are markedly conspicuous and greatly exceed in volume the loose connective tissue component of the cardinal ligament which merges into the perivesical, vesicovaginal, and rectovaginal connective tissue layers.

Figure 5 presents a sagittal section through pelvis I, 0.5 centimeter laterad to the section in Figure 3. It demonstrates the continuity of the cardinal ligament with the perivesical, vesicovaginal, and rectovaginal connective tissue layers.

Figure 5a is a high power photomicrograph of arca outlined in rectangle *A* (Fig. 5). It shows a sagittal section of the bladder and the anterior abdominal wall. Examination of the perivesical and of the subperitoneal connective tissue layer of the abdominal wall reveals identical architecture of both layers.[1]

[1]The curious irregular wavy appearance of the parietal peritoneum as well as of the bladder peritoneum is noted without venturing an explanation as to its formation and presence in the living or dead state.

Fig. 2a. *Cl*, cardinal ligament; *Co*, corpus uteri; *Pv*, perivesical layer of areolar connective tissue; *R*, rectum; *Vu*, vesicouterine pouch.

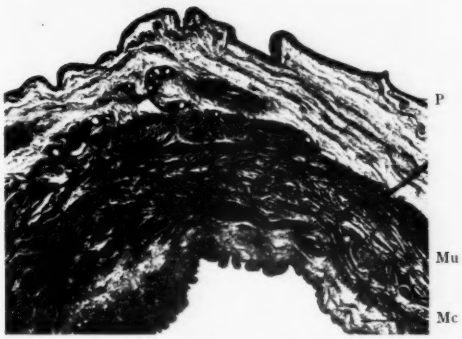


Fig. 2b. *Mc*, mucosa; *Mu*, muscularis; *P*, peritoneum.

Fig. 2c. *UB*, urinary bladder; *V*, vagina; *Vv*, vesicovaginal layer of areolar connective tissue.

Figure 5b is a high power photomicrograph of the area outlined in rectangle *B* (Fig. 5). There is a thin layer of loose connective tissue

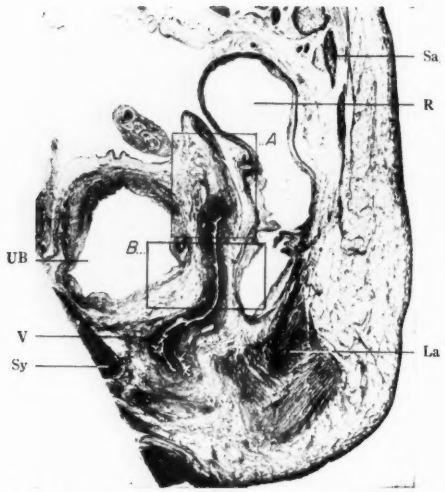
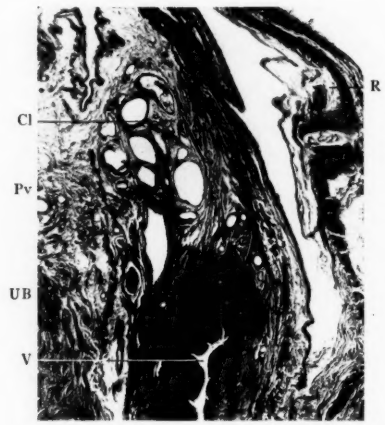


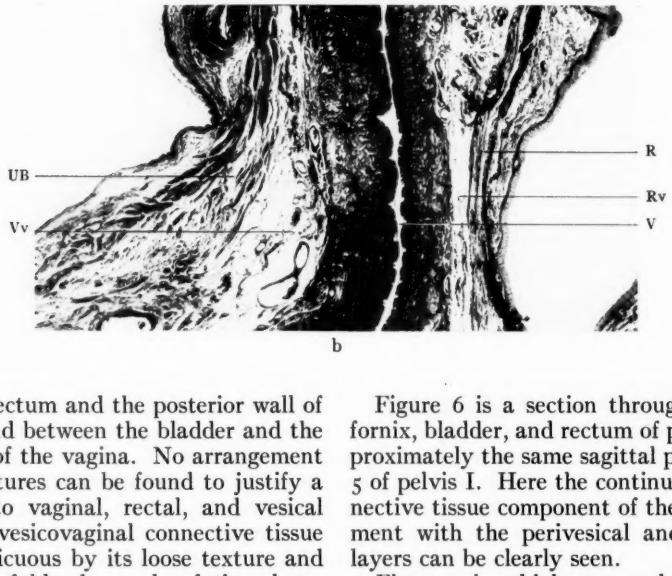
Fig. 3. *La*, levator ani muscle; *R*, rectum; *Sa*, sacrum; *Sy*, symphysis; *UB*, urinary bladder; *V*, vagina.



a

Fig. 3a. *Cl*, cardinal ligament; *Pv*, perivesical layer of areolar connective tissue; *R*, rectum; *UB*, urinary bladder; *V*, vagina.

Fig. 3b. *R*, rectum; *Rv*, rectovaginal layer of areolar connective tissue; *UB*, urinary bladder; *V*, vagina; *Vv*, vesicovaginal layer of areolar connective tissue.



b

between the rectum and the posterior wall of the vagina, and between the bladder and the anterior wall of the vagina. No arrangement of these structures can be found to justify a separation into vaginal, rectal, and vesical fasciae. The vesicovaginal connective tissue layer is conspicuous by its loose texture and the presence of blood vessels of the plexus vesicalis. The vesicovaginal and rectovaginal layers are continuous with the areolar connective tissue of the so-called cardinal ligament and of the perivesical subperitoneal layer.

Figure 6 is a section through the vaginal fornix, bladder, and rectum of pelvis II at approximately the same sagittal plane as Figure 5 of pelvis I. Here the continuity of the connective tissue component of the cardinal ligament with the perivesical and rectovaginal layers can be clearly seen.

Figure 7 is a high power photomicrograph of the cranial part of Figure 6 of pelvis II. It shows the tissue components of the cardinal ligament. The blood vessels are filled with blood as found ante mortem, and they are

surrounded by connective tissue which shows no ligamentous condensation.

Figure 8 is a sagittal section of pelvis I, outside of the lateral borders of uterus and vagina. This low power photomicrograph shows the cardinal ligament merging into the retroperitoneal connective tissue.

Figure 9 shows the cardinal ligament of pelvis I close to the pelvic wall consisting of venous plexuses embedded in loose connective tissue.

Figure 10 shows a low power photomicrograph of a coronal section through the right half of pelvis I. This section passes through uterus, vagina, and the broad ligament; the latter extends from the lateral border of the uterus to the fascia of the obturator internus muscle and continues caudad into the paravaginal connective tissue layer or paracolpos of Fothergill which fills the space between the lateral border of the vagina and the cranial fascia of the levator ani muscle. The greater part of the parametrial and paravaginal structures is taken up by blood vessels. This is clearly demonstrated since the walls of the veins of the parametrium are not collapsed, a condition commonly seen in the cadaver. In this specimen, the lumina of the blood vessels are kept open due to previous filling with latex. That the blood vessels are not overdistended by this type of preparation is readily seen by comparison with the blood vessels of pelvis II in Figures 4, 6, and 7. In pelvis II the specimen has not been subjected to any previous preparation, and the veins are seen to be filled with blood as found ante mortem. In this section of pelvis I, the abundant blood vessels of the parametrium and paracolpos are embedded in loose areolar connective tissue which continues craniad, above the innominate line, into the retroperitoneal connective tissue of the abdominal cavity. Nowhere in the broad ligament could any dense fibrous tissue be found, showing fibers grouped into large bundles characteristic of ligamentous structures. Further, it is noteworthy that the connective tissue of the parametrium and paracolpos is only in loose contact with the fasciae of obturator internus and levator ani muscles. The fasciae of these muscles, named collectively pelvic fascia, mark the boundary

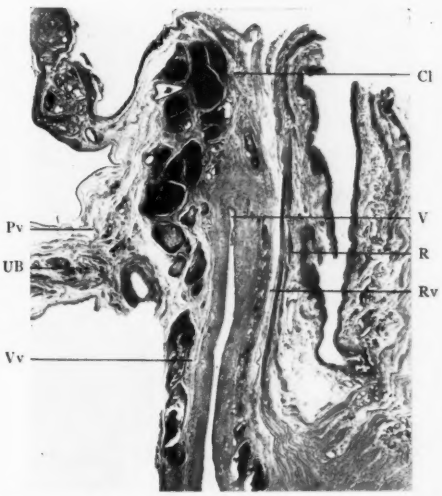


Fig. 4. *Cl*, cardinal ligament; *Pv*, perivesical layer of areolar connective tissue; *R*, rectum; *Rv*, rectovaginal layer of areolar connective tissue; *UB*, urinary bladder; *V*, vagina; *Vv*, vesicovaginal layer of areolar connective tissue.

of the pelvic wall and floor and show no direct continuation into the pelvic connective tissue.

Figure 11 represents a high power photomicrograph of a coronal section of pelvis I through the upper third of the base of the broad ligament. This photomicrograph demonstrates the abundance of blood vessels and the characteristic arrangement of loose areolar connective tissue with the wavy and irregular distribution of its fibers.

Figure 12 shows a low power photomicrograph of a coronal section of the right half of pelvis I. It passes 1.5 centimeters in front of the section presented in Figure 10. Muscle bundles of the bladder wall can be seen surrounding the ureter. This section passes close to the site where the ureter merges into the bladder. The abundance of veins in the vicinity of the ureter is conspicuous. This area corresponds to the site of the vesicouterine ligament which is a part of the pubovesicouterine ligament described in gynecologic literature. It is noteworthy that in this particular region no bandlike structure can be found. Instead there is a venous plexus, embedded in loose connective tissue which continues upward into the parametrial loose connective tissue.

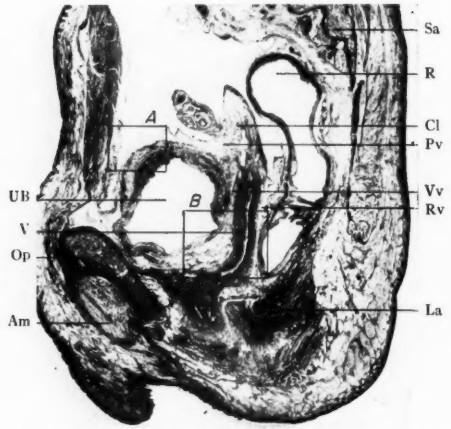


Fig. 5. *Am*, adductor muscle; *La*, levator ani muscle; *Op*, os pubis; *Pv*, perivesical layer of areolar connective tissue; *R*, rectum; *Rv*, rectovaginal layer of areolar connective tissue; *Sa*, sacrum; *UB*, urinary bladder; *V*, vagina; *Vv*, vesicovaginal layer of areolar connective tissue.

Fig. 5a. *Mu*, muscularis; *P*, peritoneum; *Pv*, perivesical layer of areolar connective tissue; *Rm*, rectus muscle; *Sup*, subperitoneal layer of areolar connective tissue.

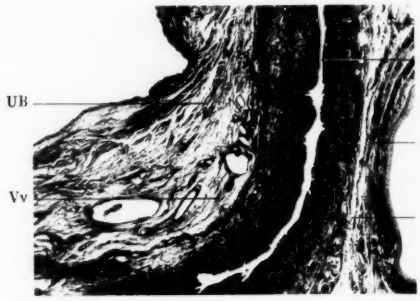


Fig. 5b. *R*, rectum; *Rv*, rectovaginal layer of areolar connective tissue; *UB*, urinary bladder; *V*, vagina; *Vv*, vesicovaginal layer of areolar connective tissue.

Figure 13 is a high power photomicrograph of a coronal section of pelvis I through the so-called vesicouterine ligament. It shows veins surrounded by loose areolar connective tissue. There is a complete absence of any ligamentous structures.

DISCUSSION

The sections of the 2 pelves that have been shown represent, in our opinion, conditions normally existing in nulliparous women.

The microscopic sections show that the subperitoneal space of the pelvis is filled with loose areolar connective tissue which is continuous with the retroperitoneal connective tissue of the abdominal cavity. There is no intimate attachment between this pelvic areolar connective tissue and the pelvic fascia covering the obturator internus and the levator ani muscles. The descriptions of a cardinal ligament as being situated in the base of the broad ligament and of a pubovesicouterine ligament as extending from the cardinal ligament to the posterior wall of the symphysis are based on gross dissections and on erroneous interpretations of the anatomic findings.

In the sections presented, it has been demonstrated that the so-called cardinal ligament is composed chiefly of the uterine venous plexus and that the vesicouterine ligament corresponds to the vesical plexus as it merges into the uterine plexus. These plexuses are embedded in loose areolar connective tissue which forms their framework.

The veins of the pelvic organs form an extensive network. The uterine plexus which communicates with the vaginal plexus is situated in the base of the broad ligament. The vesical plexus merges into the uterine plexus along the juxtavesical part of the ureter. Most of the blood of the uterine plexus is drained into the hypogastric and obturator veins. Sometimes there is also a communication of the veins of the uterine plexus with the gluteal or pudendal veins. The uterine, vaginal, and vesical arteries arise from the ventral divisions

of the hypogastric artery. The blood vessels of the pelvic organs continue into the main venous and arterial vessels situated at the lateral pelvic wall. The bulk of the visceral blood system is made up of a rich venous network. The walls of these veins are thin; in the cadaver they are generally found collapsed. In the living, the flow of blood in these plexuses apparently follows the pattern of circulation found in other plexuses in the body, that is, with only some of the pathways used under certain conditions.

On gross dissection in the cadaver, the vesicouterine and cardinal ligaments can easily be demonstrated, but they lose their ligamentous appearance completely as soon as the veins have been injected with any of the material used for demonstration of the venous system of the pelvis. When these ligaments are dissected after such injection, only abundant venous plexuses are found in contrast to the striking paucity of the connective tissue component.

To prove this observation, frequently made in our anatomic dissections, we searched for a method by which vascular and connective tissue components of these so-called ligaments could be shown intact and by which their true relationship could be demonstrated. The desired method was arrived at by filling the blood vessels to keep the lumina open, and by making histologic sections of the regions under investigation. These histologic sections show that the cardinal ligament of Kocks or ligamentum transversale colli of Mackenrodt does not exist, and that the same holds true for the pubovesicouterine ligament. Instead, blood vessels are found embedded in loose areolar connective tissue. Nowhere in these areas could any dense fibrous tissue be found with fibers grouped into large bundles as seen in ligamentous structures.

The unstriated muscle fibers not infrequently referred to as present in the parametrium can be traced in these sections as originating from the vessel walls. In the parametrial connective tissue layer there are also sparse elastic fibers present, which, however, do not exceed the amount found in the retroperitoneal connective tissue of the abdominal cavity.

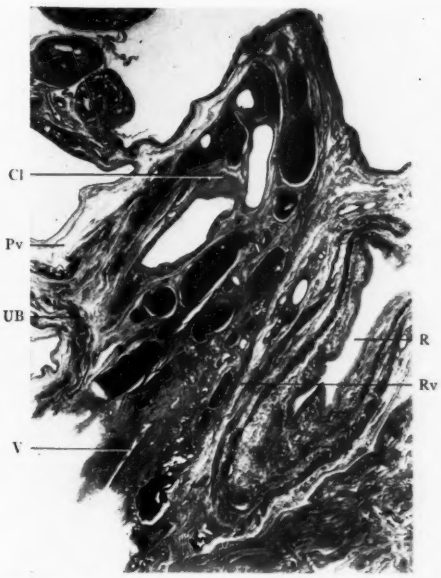


Fig. 6. *Cl*, cardinal ligament; *Pv*, perivesical layer of areolar connective tissue; *R*, rectum; *Rv*, rectovaginal layer of areolar connective tissue; *UB*, urinary bladder; *V*, vagina.

There is no tissue in the parametrium which can be termed bandlike condensations of the pelvic connective tissue. Such condensations were found only on gross anatomic dissections, especially of fixed cadaver pelves in which the tissue components were not analyzed. These condensations could be clinically confirmed on palpatory examinations alone.

During an operation for total removal of the uterus or during a Wertheim operation, it is possible to palpate, or even to have a visual impression of a bandlike condensation at the base of the parametrium. This is possible only when, during the operation, the uterus is pulled up to one side, causing traction on the parametrium of the opposite side. This traction leads to a complete compression of the lumina of the venous plexuses, their walls imparting the palpatory sensation of a bandlike condensation. It is not possible to palpate a ligament on the side which is not under tension. During vaginal hysterectomy, with the uterus pulled downward and traction exerted on the parametria of both sides, a thickened

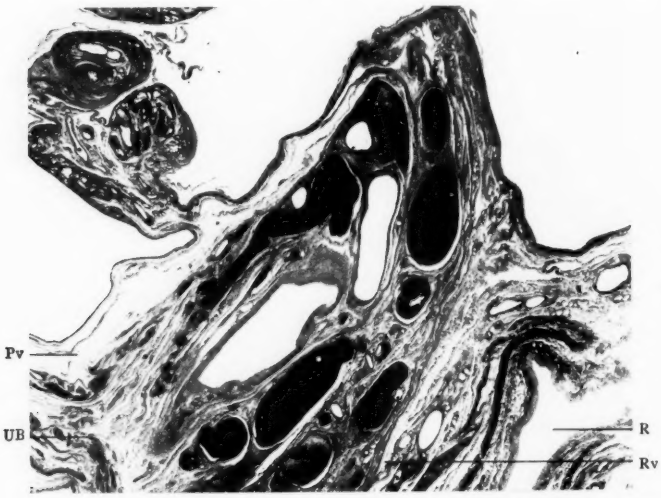


Fig. 7. *Pv*, perivesical layer of areolar connective tissue; *R*, rectum; *Rv*, rectovaginal layer of areolar connective tissue; *UB*, urinary bladder.

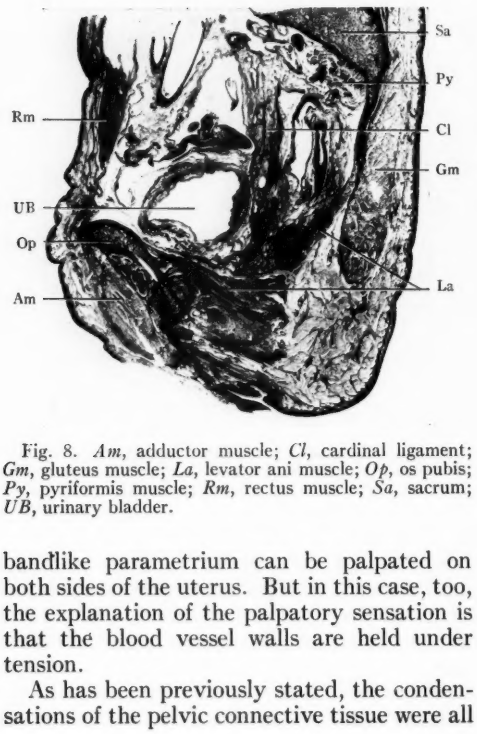


Fig. 8. *Am*, adductor muscle; *Cl*, cardinal ligament; *Gm*, gluteus muscle; *La*, levator ani muscle; *Op*, os pubis; *Py*, pyriformis muscle; *Rm*, rectus muscle; *Sa*, sacrum; *UB*, urinary bladder.

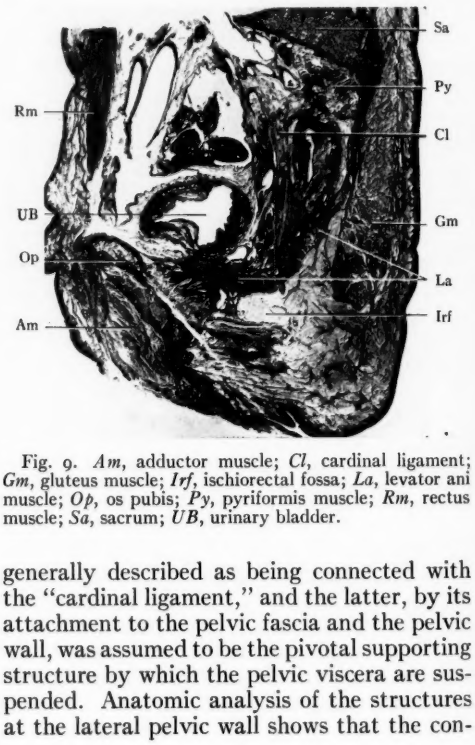


Fig. 9. *Am*, adductor muscle; *Cl*, cardinal ligament; *Gm*, gluteus muscle; *Irf*, ischiorectal fossa; *La*, levator ani muscle; *Op*, os pubis; *Py*, pyriformis muscle; *Rm*, rectus muscle; *Sa*, sacrum; *UB*, urinary bladder.

bandlike parametrium can be palpated on both sides of the uterus. But in this case, too, the explanation of the palpatory sensation is that the blood vessel walls are held under tension.

As has been previously stated, the condensations of the pelvic connective tissue were all generally described as being connected with the "cardinal ligament," and the latter, by its attachment to the pelvic fascia and the pelvic wall, was assumed to be the pivotal supporting structure by which the pelvic viscera are suspended. Anatomic analysis of the structures at the lateral pelvic wall shows that the con-

nective tissue component of the parametrium is only in loose contact with the pelvic fascia and that it is continuous with the retroperitoneal connective tissue of the abdominal cavity. There is no fixation of the parametrium to the pelvic wall through the medium of a connective tissue attachment to the pelvic fascia. The commonly assumed fixation of the parametrium to the pelvic wall can be explained by the fact that the arteries and venous plexuses, embedded in the areolar connective tissue of the parametrium, are continuous with the main venous and arterial vessels situated at the pelvic wall, as has been previously mentioned. It is obvious that blood vessels, due to their structure and physiologic function, are incapable of suspending the pelvic organs and meeting the constant changes of intra-abdominal pressure.

Legendre and Bastien, Savage, Kocks, Mackenrodt, Fothergill, and more recently Mengert, attempted in the cadaver to prove the value of the uterine supports, by exerting traction on the cervix after cutting various parts of the parametrial and the paravaginal tissues. Different degrees of uterine descent could be obtained after section of those supposedly supportive structures. A similar observation can be made in the living during vaginal hysterectomy. In this operation one can easily feel and see how the uterus gives way and comes down with each successive section of the parametrium. During these traction observations, the blood vessels and the connective tissue of the parametrium and the paracolpos prevent wide movements and displacement downward of the uterus and vagina, but this does not prove the effectiveness of these structures in maintaining the uterus in its normal position in the living female and under normal conditions. The constant changes of pressure and of traction exerted during life upon the pelvic connective tissue by the intra-abdominal pressure, affect the connective tissue if there are no other organic structures present to counteract these consequences of the action of the intra-abdominal pressure. In a defective muscular pelvic floor which leads to prolapse of the pelvic organs, the loose areolar connective tissue which has been shown in the sections to be present in the

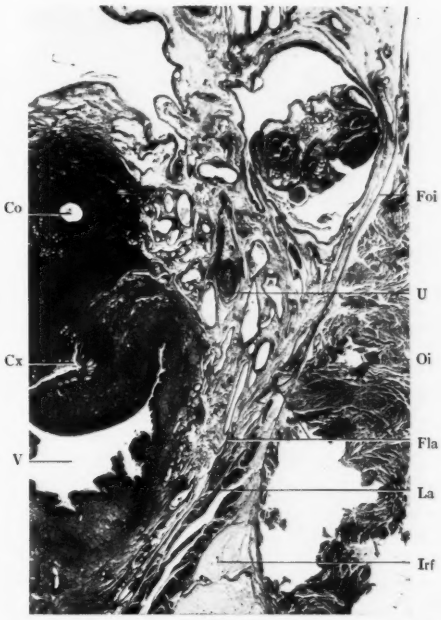


Fig. 10. *Co*, corpus uteri; *Cx*, cervis uteri; *Fla*, fascia of levator ani muscle; *Foi*, fascia of obturator internus muscle; *Irf*, ischiorectal fossa; *La*, levator ani muscle; *Oi*, obturator internus muscle; *U*, ureter; *V*, vagina.

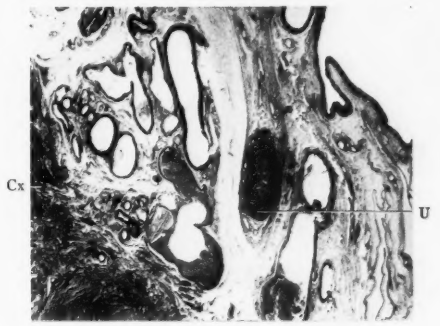


Fig. 11. *Cx*, cervix uteri; *U*, ureter.

parametrium and paracolpos instead of the bandlike condensations formerly described, cannot withstand the constant action of the intra-abdominal pressure. The reaction of the parametrium, as Berglas and others have shown, is one of elongation and hypertrophy. The parametrial and paravaginal tissues can resist the force of traction exerted on the cer-

Fig. 12. *Bwm*, muscle bundles of bladder wall; *Cx*, cervix uteri; *Fla*, fascia of levator ani muscle; *Foi*, fascia of obturator internus muscle; *Irf*, ischiorectal fossa; *La*, levator ani muscle; *Oi*, obturator internus muscle; *U*, ureter; *V*, vagina.

Fig. 13. *Bwm*, muscle bundles of bladder wall; *Cx*, cervix uteri; *Oi*, obturator internus muscle; *U*, ureter.

vix during vaginal hysterectomy and the cadaver experiments of Mengert. However, as stated before, these structures cannot withstand the prolonged and constant action of intra-abdominal pressure and, as a result, become elongated. To maintain that relaxation and elongation of the connective tissue are etiologic factors of visceral displacement is to reverse the order of cause and effect.

Another type of condensations of the pelvic connective tissue has been generally described as sheaths surrounding the pelvic organs; they have been designated as fascia of bladder, vagina, cervix, and rectum. These fasciae are said to merge with the bandlike condensations, thus forming a part of the supportive system of the viscera. Bell, Goff, Koster, and Ricci and associates in their histologic studies of the perivaginal fascia have conclusively shown that in the normal nullipara, there is no connective tissue condensation around the vagina, bladder, and rectum, which could be termed fascia. There is a thin layer of areolar connective tissue between the anterior vaginal wall and the wall of the bladder; a similar layer of areolar connective tissue separates the posterior vaginal wall from the wall of the rectum.

In the sections presented we have moreover shown that the perivesical, perivaginal, and perirectal loose areolar connective tissue layers merge with the loose areolar connective tissue of the parametrium. The names parametrial, perivesical, vesicovaginal, and rectovaginal layers serve merely to designate the topographic location and distribution of the subperitoneal pelvic connective tissue; the arrangement of the pelvic connective tissue and texture is essentially the same in these different areas. Thus there is no histologic basis for the designation of a septum vesicovaginale and rectovaginale, or a perivagina fibrosa of Halban, because these terms would imply the existence of specific structures.

CONCLUSIONS

The assumption that the pelvic connective tissue has the function of a supportive structure for the pelvic organs has been based on the supposed presence of bandlike condensations of the pelvic connective tissue, their fixation to the pelvic walls, and their attachment

to the pelvic viscera by sheathlike condensations called visceral fasciae.

Our histologic study of sections from female pelves with intact relationship of vascular and connective tissue components shows the absence of a Mackenrodt ligament and of a pubovesicouterine ligament.

The bulk of tissue structures erroneously taken for these ligaments consists of plexuses of blood vessels which are embedded in loose areolar connective tissue.

Our studies further show that sheathlike condensations of the connective tissue surrounding the pelvic organs and, designated as fasciae, do not exist.

None of the microscopic sections show attachment of the pelvic connective tissue to the pelvic walls which could be interpreted as a fixation; moreover, the pelvic connective tissue continues into the retroperitoneal connective tissue of the abdominal cavity.

There is no anatomic basis for ascribing support of the pelvic organs to connective tissue structures.

The plexuses of blood vessels and the loose areolar connective tissue are inherently incapable of supporting the pelvic organs.

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A METHOD OF RENAL RESECTION APPLICABLE TO ADVANCED HEMIHYDRONEPHROSIS AND CYSTIC HYDROCALYX

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THE laudable aim of conserving and salvaging renal tissue has resulted in the ever-increasing number of heminephrectomies for disease limited to one segment of double kidneys and of segmental resection for disease limited to one or the other pole of the unipelvic kidney.

Such conservatism can be overdone, leading to recurring illness, invalidism, prolonged periods of hospitalization, and economic disaster for the patient and his family. The highest order of good judgment must be employed in the decision to use conservative measures in these cases. Among the criteria to consider are: (1) Sufficient function must be present in the preserved organ to make the procedure worthwhile. (2) The likelihood of recurrent disease in the preserved element must be minimal. (3) The adequacy of the blood supply to the preserved element must be unquestioned since anoxic necrosis may lead not only to infection and secondary hemorrhage but also to eventual development of hypertension.

The purpose of this paper is to present a method of heminephrectomy and partial resection of the kidney applicable to certain cases, namely, advanced hemihydronephrosis and cystic hydrocalyx, which best assures the fulfillment of the third criterion mentioned.

The heretofore almost universally described technique for heminephrectomy for hemihydronephrosis consists of isolating the vascular pedicle and ligating the vessels leading to the diseased segment. This leads to blanching of the diseased segment. The line of excision is at the junction of the blanched and normal segment. The excised segment is wedge-shaped and unavoidably extends into the normal segment. The avascularity of the line of excision is purely theoretical, and hemostasis is secured by placing mattress sutures, buttressed by fat pads or muscle, through normal parenchyma and by tight suturing of preserved renal capsule over the line of excision. This latter maneuver produces hemostasis by trapping blood under sufficient pressure to equal the blood pressure within the severed vessels. The pressures being equal, the bleeding stops and clotting takes place. This technique not only includes in the excised segment a moderate amount of normal parenchyma but results in necrosis of tissue between the mattress sutures. Necrosis also results from the increased intrarenal pressure exerted by the blood trapped beneath the capsular suture line. Of equal concern is that parenchyma which has escaped complete ischemic necrosis but which has suffered permanent damage. This tissue loses its excretory function but not its endocrine function. This is the tissue responsible for the secretion of the hypertension-producing substances of renal origin.

The technique I wish to present is extremely simple (Fig. 1). It is almost completely bloodless. It does not compromise normal parenchyma. It does not endanger vascular branches entering the normal renal segment. There is no danger of perforating into a calyx of the normal segment with resultant urinary fistula. The thin-walled sac is incised and its contents aspirated. If the vascular branches entering this segment are easily observed and isolated, they are clamped, cut, and ligated. If not, the sac is incised along the convex border and the flaps then are excised along their junction with the normal parenchyma, leaving a narrow cuff. As the medial border is approached, traction on the flaps exposes the vessels very clearly, and they can be ligated without danger.

No attempt is made to excise that portion of the sac adherent to the normal segment. A

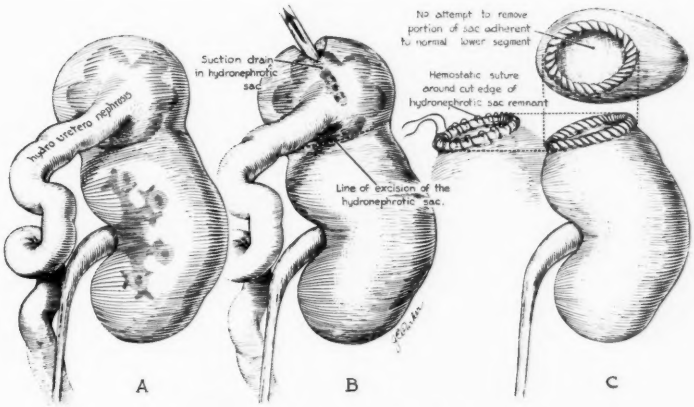
No attempt to remove portion of sac adherent to normal lower segment
Suction drain in hydronephrotic sac
Hemostatic suture around cut edge of hydronephrotic sac remnant
hydro uretero nephrosis
Line of excision of the hydronephrotic sac.
A
B
C

Fig. 1. Operation for advanced hemihydronephrosis.

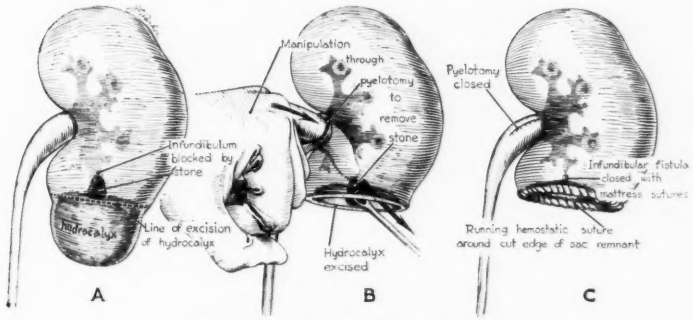

Fig. 2. Operation for cystic hydrocalyx.

running hemostatic suture of fine plain catgut is placed around the cut margin, uniting the mucosal remnant to the capsule. One then proceeds with the isolation of the pelvis and ureter, always a delicate procedure due to their adherence to their normal fellows.

The same principles are followed in the excision of the cystic hydrocalyx (Fig. 2). The thin-walled sac is excised at its junction with the normal parenchyma. A running hemostatic suture is placed along the cut margin. The patent infundibulum is closed by incising the mucosa around it and closing it with 2 or 3 horizontal mattress sutures.

CASE REPORTS

Case 1. M.H., a 6 year old girl, was referred by her pediatrician because of persistent pyuria, dysuria, and frequency unresponsive to sulfonamides. The child was known to have had pyuria for about 1 year, but the dysuria and frequency had been more pronounced during the past few months. The past medical history and family history were noncontributory. Her illness had not affected the child's general health since she was a bright and healthy-appearing little girl.

Examination of the urine showed it loaded with white blood cells and gram-negative bacilli. Physical examination was negative except for suprapubic tenderness. Intravenous pyelography showed a normal left kidney. The right renal shadow was larger than the left and showed a normal pyelographic pattern with a large amount of renal shadow above the pyelogram. This suggested a nonfunctioning upper renal segment. The cystogram showed a large filling defect of the right side of the bladder suggestive of a ureterocele (Fig. 3). Cystoscopy corroborated the massive ureterocele which made catheterization of the orifices technically impossible.

Fig. 3.

Fig. 4.

Fig. 3. Case 1. Preoperative excretory urogram. Nonfunctioning right upper renal segment and massive ureterocele.

Fig. 4. Case 1. Excretory urogram following right hemiureteronephrectomy, ureterocelectomy, and ureteroneocystostomy.

At operation, the usual flank incision was made. The kidney was exposed and was found to have a thin-walled cystic upper renal segment from which led a sausage-shaped, redundant, tortuous ureter. The operative procedure described was performed. The dilated ureter was separated with difficulty from the normal ureter as low as possible. It was then clamped, cut, and ligated, and the wound was closed without drains. The patient was then positioned on her back and a low abdominal incision was made, exposing the lower ureter extraperitoneally. The dissection from the normal ureter was very tedious and difficult. In its lower 2 centimeters it was inseparable from the normal ureter, which necessitated cutting across the normal ureter. The bladder was then opened, the ureterocele excised and its defect closed. The normal ureter was then carried through an oblique tunnel in the bladder base prepared by a curved Kelly hemostat, its end was fish-mouthed, and its flaps sutured to the mucosa by double-armed sutures which were tied on the outside. The method is that of W. W. Scott and Hugh Jewett. A No. F8 ureteral catheter was left indwelling and brought out through the urethra. The bladder was closed and drained by a Foley urethral catheter to which the ureteral catheter was anchored. One Penrose drain was directed upward, high in the flank through the lower incision, and a second one was placed near the ureteral implantation. The lower incision was then closed.

Fig. 5. Case 1. Postoperative delayed cystogram. There is no ureteral reflux.

Postoperatively, the child received antibiotics. The ureteral catheter was removed on the fourth postoperative day. The drains were shortened daily and completely removed on the seventh postoperative day. The urethral catheter was removed on the tenth postoperative day. The postoperative course was completely uneventful.

Follow-up excretory urograms at intervals over the past year show a normally functioning kidney and ureter (Figs. 4 and 5). The urine is now free of pus.

CASE 2. Mr. R.E., a 45 year old male, was referred because of persistent urgency, frequency, pyuria, and left flank pain which had been present during most of his adult life. These symptoms were associated with recurring attacks of fever and malaise which had made him a semi-invalid.

Physical examination revealed only tenderness in the left flank and left upper quadrant of the abdomen. The urine was loaded with white blood cells

Fig. 6.

Fig. 7.

Fig. 6. Case 2. Preoperative excretory urogram showing nonfunctioning left lower renal segment. Normal upper renal segment.

Fig. 7. Case 2. Retrograde ureteropyelogram fails to fill nonfunctioning lower renal segment.

and gram-negative bacilli. Excretory urograms showed a normal right kidney. Only the upper segment of an obvious double kidney filled out on the left. Cystoscopy revealed a normal bladder with only a single ureteral orifice on both sides. Retrograde pyelography, using an occlusion catheter just within the left orifice, resulted in filling only the normal upper renal segment and its ureter. Nevertheless, the diagnosis of a double left kidney with a nonfunctioning lower segment was quite evident (Figs. 6 and 7).

At operation, a cystic dilated lower renal segment was exposed. The upper renal segment was large and considered worth preserving. The ureter of the lower segment was sausage-shaped, tortuous, and redundant down to its last 2 centimeters where it became thread-like at its insertion into the midportion of its normal fellow. The operative procedure described was performed and the ureter was carefully dissected and removed down to its insertion. Closure of the wound around a single Penrose drain was then carried out.

The postoperative course was completely uneventful. The drain was removed on the fifth postoperative day and the patient was discharged on the eighth postoperative day.

Postoperative excretory urography revealed a normal left upper renal segment and ureter (Fig. 8). The patient is apyuric, free of symptoms, and working every day.

CASE 3. Mrs. E.H., a 60 year old widow, was referred because of a hard, fixed right abdominal mass, progressive asthenia and cachexia, and foul urine. Her past history revealed recurring attacks of right lower quadrant pain, fever, dysuria, and pyuria for many years.

Physical examination showed a chronically ill female with evidence of great weight loss. While lying on her back, a mass could be observed filling the right lower abdomen. On palpation, it was hard, fixed, and not tender. The urine was foul smelling and had the color and consistency of pea soup. A scout film revealed a giant renal calculus filling what appeared to be an ectopic right kidney. On the left, the pelvis was filled with many calculi in staghorn configuration. Studies of function revealed the right kidney to be completely nonfunctioning. The left kidney excreted 40 per cent of the injected phenolsulfonphthalein in 2 hours.

After correction of the patient's anemia by multiple transfusions of whole blood, the left kidney was operated upon. The pelvis and its ramifications were filled with large stones which were removed through combined pelvic and parenchymal incisions. The

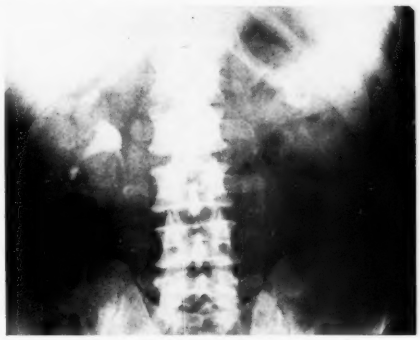

Fig. 8. Case 2. Postoperative excretory urogram.

lower pole of the kidney was a thin-walled sac equal in size to the remainder of the kidney. It was a cystic hydrocalyx which resulted from a stone impacted in its infundibulum. The thin-walled hydrocalyx was excised by scissors to its junction with the normal parenchyma and the impacted stone was removed by manipulation both through the pelvic incision and the opened hydrocalyx. The details of the operative procedure were as described before. Nephrostomy drainage was carried out. Two weeks later, a nephrectomy was performed on the other side. A solitary stone weighing 415 grams was found in this kidney.

Recovery was uneventful. There was almost no leakage from the left flank following removal of the nephrostomy tube. Postoperative pyelography of the left kidney after 2 years reveals a fairly normal-appearing, stone-free organ with good function. The patient is apyuric, has regained her weight and health, and does all the work of a busy household.

DISCUSSION

A method of surgical attack upon far advanced hemihydronephrosis and cystic hydrocalyx has been presented. There may be a reluctance to accept the procedure because the entire pathologic process is not removed; i.e., that portion of the sac adherent to the normal parenchyma. It is only necessary to remind the surgeon that incomplete removal of pathologic processes is carried out in other surgical procedures:

1. In the radical operation for hydrocele, only the parietal layer of the tunica vaginalis is removed while the visceral layer adherent to the testicle is left undisturbed.

2. Complete excisions of muellerian duct cysts are at the expense of a large part of the genital tract. Incomplete excisions, sparing the seminal vesicles, prostate, and vasa deferens, have been equally effective in obtaining cure.

3. The complete excision of serous cysts of the kidney has proved unnecessary. Merely excising the sac to the point of attachment to the renal parenchyma and placing a running suture around its edge are all that is needed. There is no need to fulgurate, phenolize, or zenkerize the serous remnant left adherent to the renal surface.

The question arises as to whether the sac remnant may secrete urine. This is most unlikely since the hydronephrotic atrophy has destroyed its functional capacity. If there were any functional elements present, the ligation of the vessels would put an end to them since there can be no secretion of urine without a blood supply. In the cases which were described, there was no postoperative urinary drainage.

SUMMARY AND CONCLUSIONS

A simple method of heminephrectomy for advanced hemihydronephrosis and of renal resection for cystic hydrocalyx is presented. The method is bloodless, rapidly performed, and free of the hazards of injury to the preserved renal segment.

OPEN, TRANSCOLONIC URETEROINTESTINAL ANASTOMOSIS

A New Approach

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THE STILL changing history of operations devised for diversion of the urinary stream to the bowel is one of the most intriguing chapters in the development of surgical technique. Hinman and Weyrauch's study of the various principles employed readily demonstrates that nearly all of them have been painfully and carefully devised with the prime aim of preventing or minimizing fecal contamination. More recently Cordonnier (2) and Nesbit have emphasized the importance of careful mucosa-to-mucosa anatomosis of ureter to bowel with attention to securing a maximal ureteral lumen without obstruction and to transplantation with minimal mobilization of the ureter. Weyrauch and Young and Woodruff and his associates have shown the desirability of securing both a good anastomosis and an adequate tunneling of the ureter between the mucosa and muscularis.

The dangers of fecal contamination have been greatly reduced by the introduction of potent antibiotics. With this advance, it seems logical to consider ureteral anastomosis under direct vision through the open bowel as it is often done in cases of reimplantation of the ureter into the bladder. Such a technique allows careful mucosa-to-mucosa anastomosis after creation of a tunnel and may be planned to employ nearly minimal mobilization of that portion of the ureter which forms the new stoma.

In 1949 and 1950 the first two authors of this paper were engaged in an experimental study of ureteral explantation and transplantation (6, 7) when the aforementioned idea gradually came to us. The technique shown in the rough sketch (Fig. 1) was tried in several dogs and 2 monkeys with only moderate success, due in part to the relative ischemia of the medial margins of the 2 ureters which were intentionally brought together in an attempt to form a sort of trigone in the bowel. Cadaver dissections, however, convinced us that the principle was fundamentally sound and simple and that it would be easier to perform in humans than in small animals. We waited for a suitable clinical opportunity to use it with the modifications shown in the later figures, where the ureteral orifices are implanted separately, yet still through the same anterior opening in the bowel wall.

This operation was successfully performed on May 7, 1952. Postoperative results have been satisfactory. The technique is presented in detail because it is believed that it would take a long time to amass a sufficient series of cases to evaluate the procedure properly in one clinic; and in the meantime the ease, simplicity, and directness of the procedure may appeal to others who might care to try it.

THE OPERATION

The illustrations demonstrate the steps of the procedure. They are drawn from below with the symphysis at the bottom and the patient's head at the top.

Preoperatively the bowel is prepared for 5 days with sulfathalidine and streptomycin (7).

A low midline incision is employed (Fig. 2). In the case reported, cystectomy and pelvic lymphadenectomy (for cancer of the bladder) were performed immediately before the transplant. The additional space in the pelvis facilitated ureterosigmoidostomy, but the opera-

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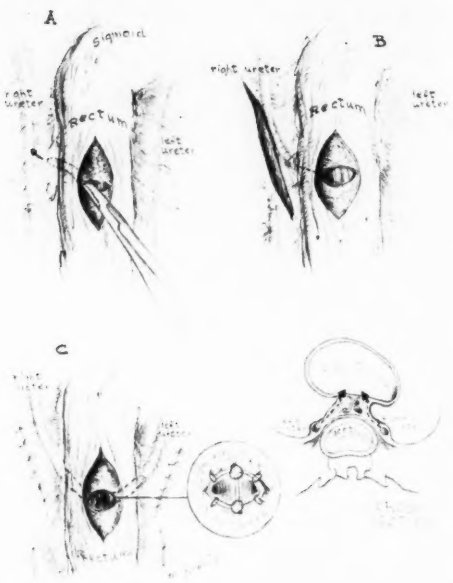

Fig. 1. Rough pencil sketch of the procedure first tried on small animals.

tion could be performed without cystectomy.

The preferred site for this transplantation, in regard to minimal mobilization of the ureters is near or below the sacral promontory where they normally are relatively near the midline in the retroperitoneal space (Fig. 3, a). The portion of the sigmoid colon overlying this area is boldly incised vertically along the taenia coli on its anterior wall for a distance of 6 to 8 centimeters. This opens the bowel and allows ample visualization of the posterior wall, which is to be the site of transplantation (Fig. 3). There is no advantage to a smaller incision in the bowel, and a smaller opening hampers later ureterointestinal anastomosis.

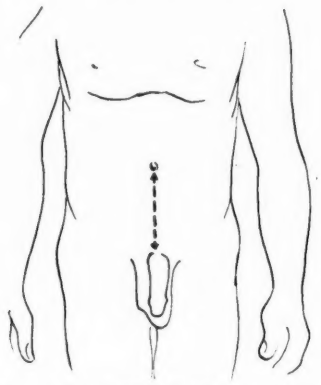

Fig. 2. A low midline incision is employed.

The ureters, which have been previously identified and severed low in the pelvis, are fixed at their free ends with traction sutures for ease in handling. The distal ends, which will later be discarded, are freed up to a point a short distance above the region where the ureter will easily and most conveniently enter the bowel from behind. Posterior peritoneum may be cut over the lower ureter to facilitate this, but the ureter remains in its normal retroperitoneal position.

An index finger is insinuated retroperitoneally and behind the bowel to push the posterior bowel wall upward (through the anterior bowel wall opening) and smooth the rugae. A 0.5 centimeter transverse incision is carefully made through the thin mucosa of the posterior bowel wall at the site desired for the position of the new ureteral ostium. A small curved clamp may then be gently introduced between the mucosa and the thin posterior bowel muscularis to create a short (1 to 1.5 cm.) tunnel upward and outward between muscularis and mucosa. When the desired tunnel length is achieved, the tip of the clamp is pointed laterally and pushed out through the muscularis into the retroperitoneal space. The jaws of the clamp are then spread to insure an adequate opening in the bowel muscle. Care is exercised to go between and not to injure the mesenteric vessels which may be palpated in this region. The tip of the curved clamp is now outside the bowel and in the retroperitoneal space near the ureter. It may be visualized by retracting the posterior peritoneum anteriorly with a deep retractor. The traction suture on the end of the ureter is seized with the clamp, and suture and attached ureter are then easily brought into the lumen of the bowel (Fig. 3). It is easier to bring both ureters into the bowel before beginning the anastomosis.

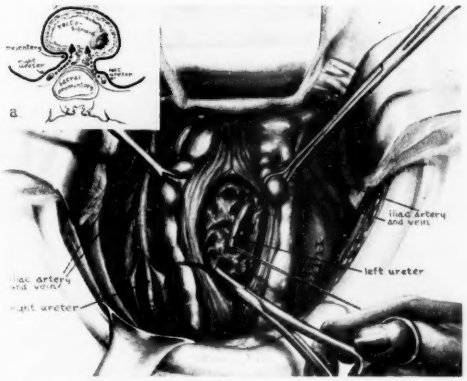

Fig. 3. In this and Figures 4 and 5 the operative field viewed from below with the symphysis in the bottom of e field. The sigmoid colon is opened widely by vertical cision of the taenia coli to expose the posterior wall of e bowel. Inset, a, shows the path that the ureters will llow when brought into the bowel from behind.

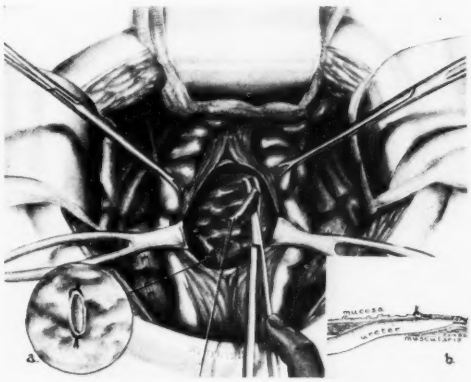

Fig. 4. Operative field from below. After incision of the bowel mucosa, the ureters are brought into the bowel through a short tunnel (insert, b) made with a small curved clamp. The distal ends are cut off and discarded, leaving the new elliptical ureteral ostium ready for anastomosis without tension.

Anastomosis of all layers of the ureter to he mucosa of the bowel is carried out with he open lumen of the ureter under direct ision. Interrupted sutures of fine chromic atgut on an atraumatic needle are used. Traction on the distal portion of the ureter ermits accurate positioning of the proposed oint of anastomosis before the distal (and schemic) end of the ureter is amputated elliptically and discarded (Fig. 4). It is of utmost importance to allow sufficient length of the ureter after amputation of the distal end to be certain that there is no tension and retraction of the new ureteral ostium. Splinting catheters led out the anus may or may not be used, depending on the operator's preference. They were used in this case because the ureters were cut a little too short, and retraction of the line of anastomosis was feared. The catheters came out spontaneously on the seventh postoperative day, and the results were quite satisfactory. It would seem that the operation should be equally successful either with or without catheters if the ureteral stump is left long enough to prevent tension and if the anastomosis is carefully performed.

When the anastomosis of the new ureteral orifice to the posterior wall of the bowel is completed, the posterior peritoneum may be closed if the surgeon wishes. It is possible, up to this time, to place an anchoring suture to hold the ureter in place outside the bowel if this seems necessary or desirable. The anterior bowel wall is then closed in 2 layers (Fig. 5). The mucosa is closed with a continuous suture of fine catgut, and the muscularis is approximated over this suture line with interrupted black silk sutures. No intraperitoneal drains are used.

After operation the rectum is drained of urine continuously with a large mushroom

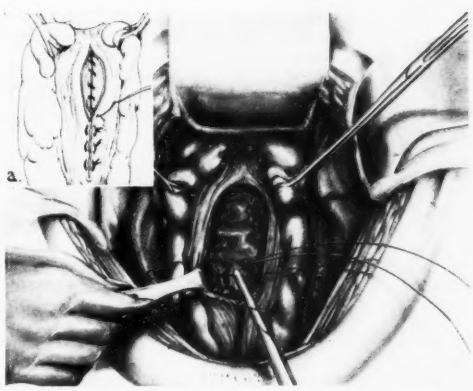

Fig. 5. Operative field from below. Direct, elliptical anastomosis of ureter to bowel mucosa is performed with interrupted fine catgut sutures. The colotomy opening is closed in two layers.

Fig. 6.

Fig. 7.

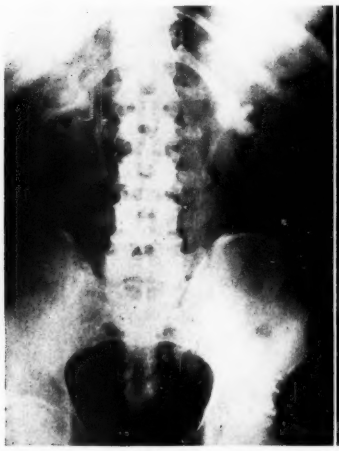

Fig. 8.

Fig. 9.

Fig. 6. Preoperative intravenous pyelogram, April 28, 1952.
Fig. 7. Intravenous urogram 3 weeks after operation, May 26, 1952.
Fig. 8. Intravenous urogram 2 months after operation, July 14, 1952.
Fig. 9. Intravenous urogram 11 months after operation, April 14, 1953.

catheter. Antibiotics are used freely and fluid and electrolyte balance are carefully observed and maintained.

RESULTS

The early results in the case reported herein have been satisfactory when compared with results obtained by other methods. There have been minimal electrolyte changes. Postoperative intravenous pyelograms compare favorably with those before operation (Figs. 6, 7, 8, 9). Figure 6 is a preoperative excretory urogram made on April 28, 1952. The operation was performed May 7, 1952. Figure 7 (May 26, 1952) shows the appearance 3 weeks after surgery, and Figures 8 and 9 show follow-up

urograms made on July 14, 1952 and April 14, 1953.

We have been encouraged by the results obtained in this patient and plan to employ the method in suitable cases in the future.

DISCUSSION OF ADVANTAGES AND POSSIBILITIES

The theoretical advantages of this simple method are appealing.

The operation, as outlined, resembles reimplantation of the ureter into the bladder, which is a procedure familiar to all urologists.[1] Emphasis here has been placed on the desirability of careful mucosa-to-mucosa anastomosis; however, it may well develop that the even simpler use of long intraluminal flaps of ureter, attached to the bowel wall, as advocated by Stevens and Marshall for ureteroneocystostomy may be a satisfactory variant of this method.

The elliptical anastomosis of ureter and its mucosa to the mucosa of the bowel may be performed carefully and perfectly under direct vision, insuring an open, functional stoma. This type of anastomosis has been described as the method of choice by Sayegh in his study of reimplantation of the ureter into the bladder for stricture.

The ureters enter the bowel posteriorly from behind the peritoneum where they normally lie quite close to the midline near the brim of the pelvis and just below the iliac vessels, the optimum site for preserving blood supply of the human ureter as demonstrated by Daniel and Shackman. This provides minimal handling and deviation of the ureters from their normal course, and helps to insure an adequate blood supply of the portion of the ureter which is used to create the new ureteral orifice.

The site of anastomosis of ureter to bowel is, by the nature of the operation, retroperitoneal and low in the bowel (Fig. 10).

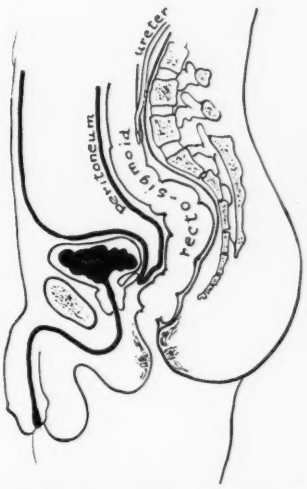

Fig. 10. Parasagittal section diagrammatically represented to show retroperitoneal site of ureteral transplant near the normal bed of the ureter.

The procedure combines the advantages of a short tunnel and a direct elliptical anastomosis. The possible valve-like action of the bowel wall, pressing on the ureter as it lies in front of the sacrum, is theoretically likely to be more efficient.

Two layer closure of the anterior colotomy, employed in this procedure, is simple and should not add to the dangers inherent in ureterointestinal anastomosis by any route. The idea of opening the bowel widely in order to achieve a desired end should not be alarming to urologists who heretofore usually have traditionally avoided it. Colotomy in order to revise an improperly functioning ureterosigmoidostomy stoma has been reported favorably by Henderson and others.

This type of ureterointestinal anastomosis may prove to be particularly applicable in cases in which a substitute bladder is created after radical pelvic surgery. It would seem that an isolated loop of bowel, open at both ends might lend itself especially to this type of open anastomosis under direct vision before closure is performed.

Cordonnier (3), Boyce and Vest, and more recently Creevy and Reiser have expressed the cogent thought that perhaps patients who

[1]Dr. Wilson Stegeman has independently conceived and performed this operation. (*Transactions Western Section American Urological Association, 1952*). He reports in personal communication that he has performed 2 such operations and continues to be enthusiastic about the method of transcolonic anastomosis and expects to use it exclusively in the future. In his first case, a palliative one, the patient died from uremia because of an undiagnosed obstructing blood clot in the only functioning kidney. There was no peritonitis at autopsy. The second patient is doing well.

Dr. Leander Riba (J. Urol., Balt., 1952, 67: 284, Fig. 1) has used an open method of ureterointestinal anastomosis as the second part of a two stage operation in animals.

must suffer diversion of the urinary stream into the lower bowel should also have a proximal diverting colostomy to protect the urinary tract from infection and its sequelae. The operation of open ureterointestinal anastomosis described in this article would be ideally suited to such a procedure.

SUMMARY AND CONCLUSIONS

A new approach to the problem of ureterosigmoidostomy is presented. The technique is described and illustrated in detail.

The anterior wall of the sigmoid colon is opened in the region of the sacral promontory. Through this colotomy the ureters are anastomosed to the posterior bowel wall very much as in the operation of reimplanting the ureter into the bladder.

Direct mucosa-to-mucosa anastomosis of elliptical ureteral stoma to bowel is achieved with the site of transplant under vision. A short tunnel is created, and there is minimal deviation of the ureters from their normal course and blood supply.

An illustrative case is briefly presented. Theoretical advantages and possibilities of the procedure are discussed. The approach is worthy of further consideration and trial.

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INDICATIONS FOR SURGICAL TREATMENT OF COARCTATION OF THE AORTA

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THE SURGICAL treatment of coarctation of the aorta was demonstrated to be feasible by Crafoord and Gross. The comprehensive studies of Abbott, Blackford, and Reifenstein, Levine, and Gross established that this anomaly was associated with high morbidity and decreased life expectancy, making serious consideration of these newly developed surgical techniques mandatory in the treatment of coarctation of the aorta. In the last 4 years in this clinic, 22 patients have been operated upon for coarctation of the aorta. In this group there were 4 instances of cardiac failure, 6 instances of aortic regurgitation, and 1 of mitral stenosis. Since the indication for treatment of coarctation of the aorta when associated with other anomalies is not clear, our experience in this group of cases is presented.

MATERIAL

From January 1, 1948 until September, 1952 there have been 28 patients with the diagnosis of coarctation of the aorta observed by the Thoracic Surgical Division of Duke Hospital. Two patients, both over 18 years of age, refused operation. A young girl of 4 years of age was not offered operation because of marked cerebral degeneration from repeated cerebral hemorrhages. There were 2 young men who died from subarachnoid hemorrhages before surgery could be done. In 1 of the patients operation had been refused 3 years before. The other patient had been explored at another clinic a year before death, but excision of the stenotic aorta had been deferred because a graft was not available. Two infants with coarctation are now being followed with the hope that operation can be deferred until they are older.

Exploration has been carried out in 22 cases of coarctation of the aorta, and in all the obstruction was relieved. The ages of the patients ranged from 16 months to 31 years, with 5 under the age of 14. Twenty patients had an end-to-end anastomosis while 2 had aorta to subclavian anastomosis. The only postoperative complication that occurred in a patient who survived was a subarachnoid hemorrhage on the fourth postoperative day.

In the first 19 cases there were no deaths. The twentieth case was of a 22 year old male who had complete aortic stenosis with tremendously enlarged collateral vessels. The aorta was mobilized with difficulty, but finally after resection an entirely satisfactory union was obtained. His postoperative period was uneventful until the seventeenth postoperative day when he developed sudden pain in the left posterior chest followed by circulatory collapse. The patient was carried to the operating room where the left chest was opened. This had to be done very quickly as the patient's heart stopped beating as soon as he was placed in the lateral position. By the rapid transfusion of blood and cardiac massage the patient was revived. Just above the suture line there was a perforation in the aortic wall. This was easily repaired with interrupted sutures. Following the second operation the patient did well until the sixteenth day when again aortic rupture occurred. This time the repair was not successful, and the patient died. It was evident at the second operation that a large area of aortic arch in the region of its junction with the left subclavian artery was necrotic. There was no evidence of bacterial infection about the suture line.

The twenty-first patient, a male, 26 years of age, had been in heart failure on admission to the hospital and, in addition to coarctation, had severe aortic regurgitation and mitral stenosis. After adequate preoperative preparation, a satisfactory repair of a simple stenosis was effected. On the sixth day after operation

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the patient developed abdominal pain. On the tenth postoperative day marked abdominal distention occurred. It was thought that the patient had mesenteric thrombosis, and exploration of the abdomen was done. The jejunum and first portion of the ileum were found to be edematous and markedly cyanotic, but the superior mesenteric artery and its subdivisions pulsated. The mesenteric veins were patent. Death occurred while exploration was in progress. The autopsy revealed inflammation of the small arteries and arterioles confined to the body area supplied by the aorta below the coarctation. There were numerous infarcts in the liver, spleen, kidneys, and intestines. There were no emboli to the mesenteric arteries or thrombi in the mesenteric veins. The suture line in the aorta was healed, and thrombi were not present either in this location or on the scarred mitral valve. A more detailed report of this case is to appear at a later date.

In this group of patients with coarctation, aortic regurgitation was suspected in 6 cases and was associated with cardiac failure in 3 of them. The combination of aortic regurgitation and stricture would seem to place an undue stress on the heart, for by x-ray examination 5 of these 6 patients were seen to have cardiac enlargement. There were 5 instances of cardiac enlargement not associated with aortic insufficiency. In the 10 patients with enlarged hearts who survived, there was a significant reduction in the size of the heart after operation.

Previous cardiac decompensation had been present in 4 cases of this series. In all, the failure was of such intensity that control by medical management was difficult and unsatisfactory. The first case was that of a man of 31 years of age who was well except for known hypertension of 5 years' duration. He had few symptoms until 2 years before admission when he developed a marked increase in his dyspnea. Nine months before operation heart failure occurred, necessitating 2 hospital admissions and strict medical supervision for control. The diagnosis of coarctation of the aorta and aortic insufficiency was made. Operation was finally decided upon in spite of his poor cardiac reserve, age, and the presence of aortic regurgitation. A subclavian-aortic anastomosis had to be done because of a long aortic stricture. Following operation he has shown marked improvement (Fig. 1). Now, 4 years later, he works every day and does not require any treatment for his heart. The patient still has a murmur and high pulse pressure diagnostic of aortic insufficiency, though his brachial systolic pressure is reduced from 180 to 150 millimeters of mercury.

The second case of heart failure occurring before operation was that of an 8 year old boy who had been under treatment for cardiac failure for 3 years. He had a greatly enlarged heart, a mitral diastolic murmur, hypertension of 160 to 180/90, and a greatly enlarged left auricle. The diagnosis of mitral stenosis, as well as coarctation of the aorta, was made. Repair of the coarctation was done without incident, and since operation there has been a dramatic improvement in his cardiac reserve. Now, 2 years later, he no longer requires digitalis or salt restriction and has practically no limitation in his physical activity. There has been some decrease in heart size (Fig. 2), but there is still clinical evidence of mitral stenosis.

The third case was of a 16 month old infant who had had difficulty maintaining adequate circulation since 7 days of age, in spite of digitalis and careful supervision. End-to-end repair of the coarctation was done. Improvement has been striking in this child with accelerated development, until 2 years after operation the patient is of normal size for her age. The cardiac size decreased almost to normal. Since operation the child has had a persistent high pulse pressure (160/80) and is suspected of having an incompetent aortic valve.

The fourth case in the failure group was of a 25 year old colored male with cardiac failure and severe aortic regurgitation. This patient did not survive, and his course following operation has already been outlined. His death was not related to cardiac failure but to a peculiar thrombosing vascular disease confined to the body area below the coarctation.

In 17 of the cases in this series an end-to-end anastomosis of the aorta was carried out, while in 2 cases an anastomosis of the left sub-

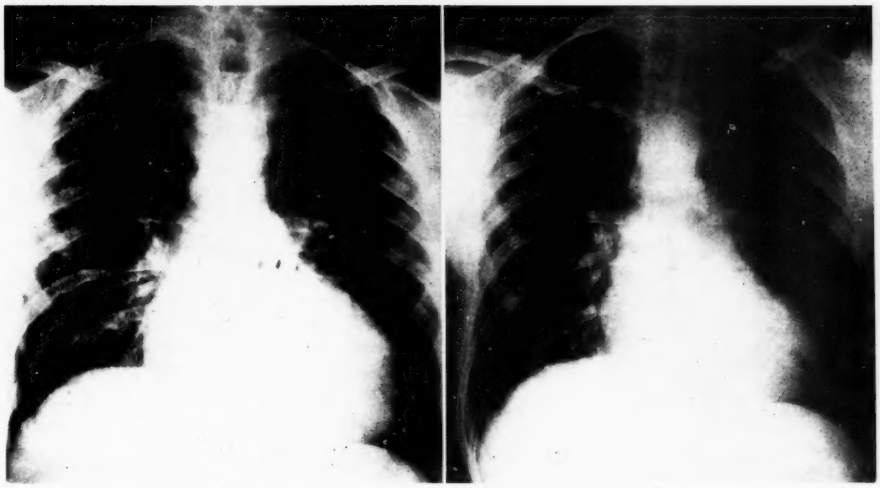

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Fig. 1. a, Preoperative and, b, postoperative chest plates of a 31 year old patient with coarctation of the aorta associated with aortic insufficiency.

clavian artery to the distal aorta was made. In the cases of heart failure outlined previously, there was 1 patient in whom this latter procedure was performed with dramatic results. The second patient subjected to this same procedure also obtained relief of his symptoms. The pressure in his arms changed very little, though the pressure did increase in the lower extremities (from 90 systolic to 130 to 140 systolic).

In 19 of the 22 cases followed after operation for coarctation of the aorta there has been a reduction in the systolic pressure to normal. In 1 of the failures a subclavian-aortic anastomosis had been performed, and though his brachial pressure (180 systolic) did not decrease, the pulsations and femoral pressure did increase (0 to 120). There was, in a second patient with aortic regurgitation, a failure to reduce the systolic pressure to normal, though the blood pressure in the lower extremity has increased so that there is, by cuff measurement, the normal differential between the arms and legs. The third patient, 16 months of age at the time of operation, still has a high systolic pressure and is suspected of having an incompetent aortic valve, though all symptoms of cardiac failure have been relieved.

In the group operated upon there were 2 patients who had had previous cerebral accidents, and they were left with marked residual neurologic deficits. The episodes had occurred 2 years and 11 years before surgery. Since operation there has been no recurrence after 1 and 4 years' time respectively. They were aged 15 and 17 years at the time of their cerebral accidents. One patient had a subarachnoid hemorrhage 4 days after operation. There were 3 instances of subarachnoid hemorrhages in the group of patients who were not operated upon, giving a total of 6 cases of intracranial bleeding in 28 instances of coarctation of the aorta.

DISCUSSION

The decision to subject a patient who has few or no symptoms to such a formidable operation as resection and repair of the thoracic aorta should be based on solid indications. Reifenstein, Levine, and Gross have investigated this aspect of the problem; and their study of the patients over the age of 2 who had died from coarctation of the aorta indicated that the life expectancy was shortened. Of these patients roughly one-fourth died from rupture of the aorta; one-fourth from endoarteritis or endocarditis that in most instances

was on the aortic valve area or adjacent to the strictured area; one-fourth from cerebrovascular accidents or cardiovascular failure; while the remainder succumbed to causes not related to the vascular system. It must follow then that in one-fourth to one-half of the cases, according to these criteria, death could be attributed to changes occurring in the region of the deformed aorta, that is, vascular rupture and in some instances arterial infection. In our 22 cases, when the indications for operation were appraised, 10 patients were found to have been operated upon primarily to prevent these latter accidents from occurring.

The eventual development of severe generalized cardiovascular disease as a result of coarctation of the aorta is still open to question. In this study there were 5 patients with enlarged hearts who had only coarctation. It is difficult to estimate how many of this group would have gone on to failure or severe generalized vascular disease. In the younger age group, as in 3 of these 5 who were below 9 years of age, appreciable cardiac enlargement should always warrant surgical intervention. Slight enlargement when the patient is above the age of 30 might alone be of questionable significance in determining the need of surgical intervention. Even though studies (1, 7) already quoted indicate that one-fourth of the patients succumb to cardiovascular disease, this includes intracerebral bleeding from aneurysms as well as the group who have associated cardiovascular anomalies or diseases.

When coarctation of the aorta is associated with another cardiac anomaly or disease, the additive effect of the two may be such that failure of the circulation is likely to occur. In this series there were 6 cases of aortic insufficiency and 1 of mitral disease. Four of these 7 patients had heart failure. Reifenstein, Levine, and Gross could find in their study just 1 case of heart failure in patients over the age of 2 years in which coarctation alone was present. The observation that associated defects in the heart add to the deleterious effect of coarctation was borne out by Calodney and Carson's study in infants dying with coarctation of the aorta. In 21 autopsied patients in their study, 12 were considered to have minor associated cardiac defects, and these included bicuspid valves, interventricular defects, hypoplasia of the left ventricle, absent right coronary artery, atresia of the aortic arch, and hypoplasia of the right ventricle with aplasia of the tricuspid valve. There were only 4 cases of failure when there were no abnormalities noted other than the coarctation. Olney and Stephens report 5 patients under the age of 4½ years, all of whom had had cardiac failure. Two of this group had mitral stenosis. The experience in our series of cases as well indicates that coarctation of the aorta, when associated with other defects as aortic regurgitation or mitral stenosis, constitutes the most urgent indication for surgical intervention.

The occurrence of intracranial bleeding in coarctation of the aorta presents an interesting problem and enters into any evaluation of the indications and the results of operation. Cerebral aneurysms and coarctation are two associated defects, and it is not illogical to assume that the hypertension occurring with the coarctation, particularly the very high pressures associated with stress, might make the aneurysm more likely to rupture. On the other hand, normotensive patients with intracerebral aneurysms succumb to this accident with the same frequency as do hypertensive patients, and after the first hemorrhage the mortality rate in all untreated cases is 90 per cent. It is perhaps incorrect to assume that repair of the coarctation alone might result in any significant change in the prognosis when cerebral aneurysms are associated with coarctation.

The influence of the age of the patient on the indications for operation in coarctation of the aorta is considerable. In the very young, operation is mandatory, when adequate circulation cannot be maintained and development is retarded. When the patients are 5 to 10 years of age, there are technical advantages in performing the operation at this time that outweigh any consideration of further growth at the anastomotic site. When the patient is above the age of 30, the operation becomes more hazardous. The mere diagnosis of coarctation associated with mild

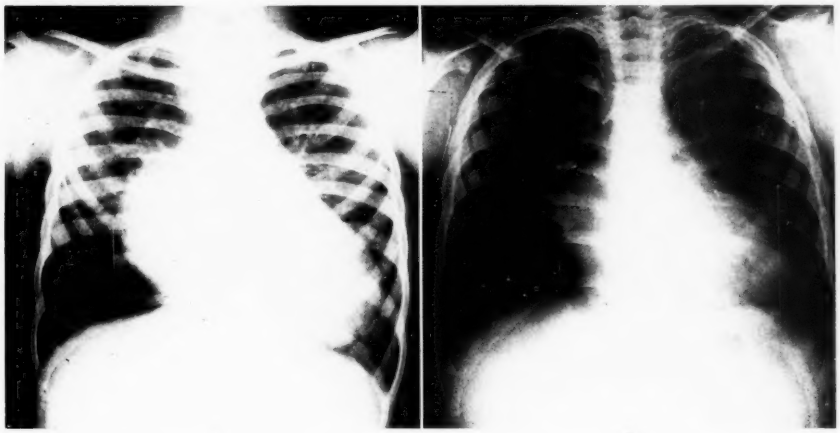

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Fig. 2. a, Preoperative and, b, postoperative chest plates of an 8 year old patient with coarctation of the aorta associated with mitral stenosis.

systolic hypertension does not constitute an unequivocal indication for operation in this age group.

In the presence of a long stricture the advisability of doing an aortic-subclavian anastomosis is open to question at the present time. In the 2 cases cited here, the results have been striking in one and only partially satisfactory in the other. The decision in most instances of whether to use this procedure or to insert a graft would seem to be dependent upon the indication in the individual patient for the operation. As would be the case in 25 to 50 per cent of cases, the operation is for the prevention of rupture or infection in the region of the coarctation; it is questionable if one should substitute a homologous aortic graft for a strictured viable aorta. If the lumen of the aorta can be increased appreciably by an aortic-subclavian anastomosis, one gets a tremendous increase in the blood flow through the operative site; and this was shown by the relief of failure of the heart in one of our cases and objective evidence of increase in circulation in the lower extremities in the other. By increasing the size of the lumen and causing the blood flow at the anastomotic site to increase, the size of the large collaterals should decrease. This would remove much of the turbulence occurring just below the stricture, and the trauma to the aortic wall would be decreased, therefore reducing the chances of infection and rupture. In addition, this would prevent, by slowing collateral flow, the possibility of aneurysmal formation in the large intercostals. In instances in which there is an aneurysm of the aortic wall or advanced sclerotic disease of the aorta in the region of the stricture, a graft would be mandatory as a life-saving measure, providing, of course, the left subclavian was diseased or was not long enough to bridge the defect.

In evaluating the results of the operation for coarctation of the aorta, a fall in the systolic pressure to normal levels gives the best indication of a successful repair and restoration of an adequate aortic lumen. On the other hand, this should not be the sole criterion for evaluation, as the persistence of a supranormal systolic pressure in the upper extremity after operation does not necessarily imply that the flow through the repaired aortic segment is not greatly increased as has already been emphasized. Unfortunately there is no objective way of determining the residual abnormal stress on the vascular wall at the site of the anastomosis when there is not complete restoration of aortic lumen to normal size. This reduction in the stress as well as the removal of the necessity for a large volume of blood to pass through collaterals is important in assessing the result of operation.

CONCLUSION

In nearly half of the cases of coarctation of the aorta, operation is indicated to prevent the occurrence of accidents or infection in the region of the coarctation.

Coarctation of the aorta when complicated by some other anomaly of the heart or great vessels presents great risk of heart failure. In instances of congestive failure when a coarctation of the aorta is associated with another defect, the removal of the coarctation will improve cardiac function.

The combination of coarctation of the aorta and some other anomaly of the heart or great vessels constitutes the strongest indication for surgery.

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CONGENITAL MEDIAN BAND OF THE ANUS

A Report of Six Cases with Results of Surgical Treatment and a Discussion of Possible Embryologic Events Leading to the Abnormality

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AMONG the congenital abnormalities of the anus there is one condition which we believe has been frequently overlooked, or else has been confused with imperforate anus. This condition is characterized by the existence of a band of tissue running in an anteroposterior direction across the anus. The band is composed of connective tissue and fat, covered by skin. The true nature of the condition can frequently be discovered only by close inspection.

In a survey of 68 articles dealing with congenital abnormalities of the anus we have been able to find but 4 similar cases recorded (2 cases by Browne and 1 each by Drueck and Saphir). We wish to add the descriptions of 6 cases encountered during the past 3 years.

CASE 1. K.B., a 2 month old male infant, was first seen in September of 1950. He had been severely constipated since birth. Examination showed a band of tissue running in an anteroposterior direction across the anus, with two tiny openings, one on each side. At operation this was excised. It was also found that the anus was stenotic. This was dilated digitally, and the infant has had normal rectal control since that time.

CASE 2. T.W., (case of Dr. Donald T. Hall), a male, aged 1 year, was first admitted to the Children's Orthopedic Hospital in 1947. At that time the baby was described as having a "stenotic, imperforate anus with 2 fecal fistulas." The child had always been constipated. A small amount of barium injected through one of these fistulas showed what appeared to be a relatively long area of stenosis extending upward from the anus. Because of the x-ray findings a colostomy was done and the child was allowed to develop until October of 1950, at which time an anoplasty was performed. At operation it was found that the anus was bridged by a thick anteroposterior band of tissue, and that above this there was a short stenosis of the anal canal which could be dilated. Following closure of the colostomy this child has had entirely normal bowel movements, and his anus has a normal appearance and tone.

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CASE 3. S.K., a 1 day old male, was admitted to the Children's Orthopedic Hospital in December 1951 with a diagnosis of imperforate anus. Close examination, however, showed 2 tiny dimples, one on either side of a median bridge lying inside the anal pit. Because of our previous experience we thought that this case might be similar to those described. At the end of 24 hours meconium appeared through one of the openings at one side of the anus. We then found that we could easily dilate the anus through this opening. This child has been having normal bowel movements since that time. The band of tissue has been left in place at one side of the anus but can be removed later if desired.

CASE 4. D.C., a 3 hour old male infant, was seen in December of 1949 with a diagnosis of imperforate anus. Examination revealed the following: There was a band of tissue in an anteroposterior direction across the anus with a tiny fistula passing through the middle and a very thin membranous diaphragm on either side of it. This band was excised without anesthesia. The sphincter which was tight, as in the other cases, was dilated leaving an entirely normal anus. The baby has been well since that time.

CASE 5. Von B., a male, aged 7 months, was seen in July of 1951. Examination of the perineum revealed findings as shown in Figure 3. The fecal stream was passed through a perineal opening beneath a bar of tissue which extended from the posterior margin of the scrotum across this opening and continued posteriorly across an imperforate anal dimple. Thus the condition involved both a congenital median band and a rectoperineal fistula. The treatment consisted of first excising the band and then, at a second operation, transplanting the perineal fistula back into the normal anal musculature. A follow-up visit 6 months after the second operation showed that this baby had normal anal control and a normal appearing anus.

CASE 6. W.M., a 6 month old male, was first seen by us in 1949. At birth the baby had been seen by his own physician, who stated that he found a band of tissue placed in an anteroposterior direction across the anus with a thin diaphragm on each side of it which he perforated. At 2 months the baby began to have urinary infection. When we saw him at the age of 6 months, there was an extremely tight stenosis of the anus together with an anteroposterior band of tissue along one side of the outlet, and an associated rectourethral fistula. By x-ray examination the stenosis was apparently quite long, as in Case 2, and

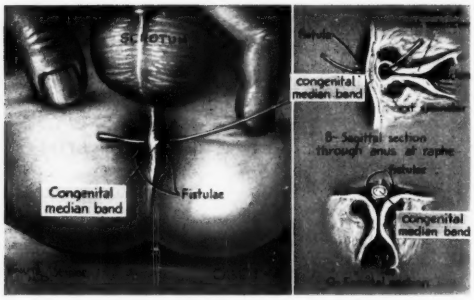

Fig. 1. Congenital median band of the anus in 2 month old infant.

a colostomy was accordingly performed. At the age of 2 years he was operated on through a perineal approach and the stenosis proved to be limited to the anus itself. The stenotic anus was enlarged and the rectourethral fistula was divided. He is now, we hope, developing anal control although the follow-up is too short to be certain.

EMBRYOLOGY

The primitive cloaca is a common vestibule formed by the dilated end of the hindgut and entered also by the allantois and by the right and left mesonephric ducts. It subsequently becomes divided into two parts by the caudal growth of the urorectal septum which descends between the allantois and the hindgut. The cloaca is thus divided into the ventrally placed urogenital sinus and the primitive rectum, dorsally.

A cloacal membrane formed of endoderm internally and ectoderm externally forms a covering which separates the cloaca from the exterior. Seen from the exterior in the intact embryo, the cloacal membrane is at the bottom of a depressed area or furrow, the genital fossa or external cloaca (Fig. 4A).

As the urorectal septum grows caudalward the cloacal membrane disintegrates, apparently by desquamation, and permits both the hindgut and the urogenital sinus to open into the genital fossa and thus to the exterior. The more dorsal part of the cloacal membrane lies below the rectal tube and the membrane may briefly occlude the rectal tube when the urorectal septum descends to reach the membrane. This portion of the membrane may persist for a time after the more ventral part, which is covering the urogenital sinus, has disintegrated. This persistence of the posterior part has led to the concept of the anal membrane or plate. It will be seen that it is but a temporary persistence of the cloacal membrane at the lower end of the rectal tube.

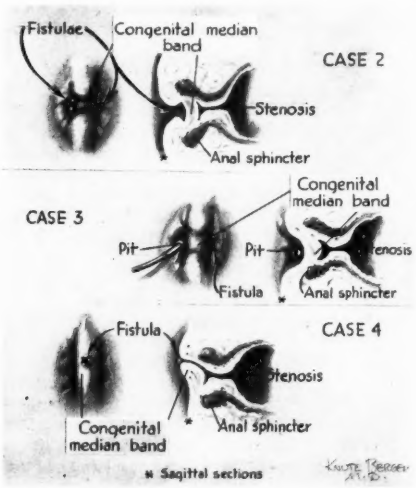

Fig. 2. Congenital median band of the anus as seen in 3 infants: Case 2, aged 1 year. Case 3, aged 1 day. Case 4, aged 1 day.

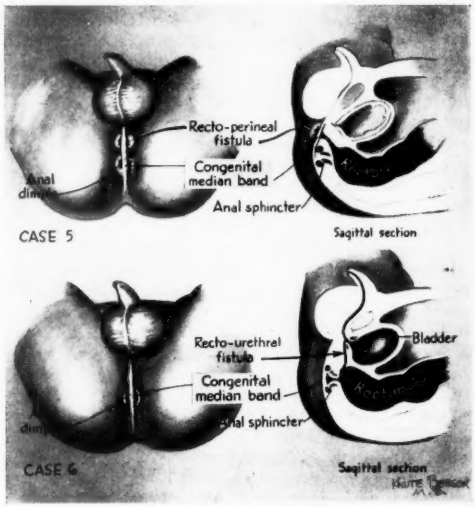

Fig. 3. Congenital median band of the anus which was associated with other abnormalities as seen in 2 infants, Cases 5 and 6.

Tench has pointed out that this membrane will usually have entirely disappeared by the time the embryo has reached 22 millimeters in length.

The termination of the hindgut is now formed by the rectum becoming continuous with the external cloaca along its lateral and posterior walls, while the anterior wall of this extended lower end of the hindgut is produced by the lower end of the urorectal septum. This terminal part of the hindgut is to become the upper part of the anal canal, for the anus itself is yet to be formed by more superficially placed tissues.

Prior to the disintegration of the cloacal membrane a slight eminence forms on either side of the midline dorsal to the site of the future anus and just ventral to the tail. These are the anal tubercles described by Reichel in 1888 (Fig. 4A). The anal tubercles appear to be connected to one another by a slight elevation of tissue known as the transverse fold or band. This transverse median band plus the anal tubercles on either side form the boundary of the most dorsal portion of the genital fossa.

The anal tubercles next grow forward or ventralward in such a manner as to form a horseshoe-shaped elevation (Fig. 4B). The center of the concavity of this curved elevation is the site of the future anal aperture, for this C-shaped elevation is encircling the termination of the hindgut. The anal tubercles continue to expand ventralward and medialward until they converge in the median plane, completing the anterior margin of the anus by their final union here (Fig. 4C). The right and left margins of the external cloaca immediately adjacent to the converging anal tubercles also fuse in the midline, and thus the central tendinous area, or perineal body, of the definitive specimen is formed (Fig. 4B). The anal tubercles by their growth ventralward and final convergence and by their connecting median band dorsally have thus created a circular elevation with a central dimple. This is the anal mound and the central depression (proctodeum) is the outlet of the hindgut (Fig. 4D). At least part of the substance of the anal mound is produced by the developing sphincter ani externus muscle. Popowsky showed

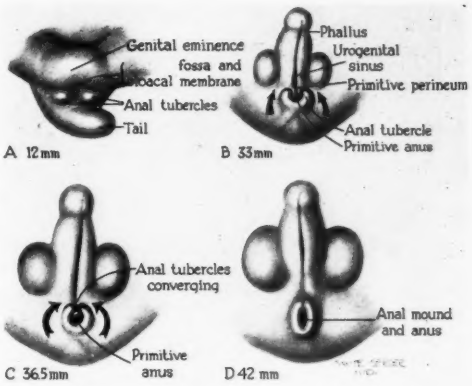

Fig. 4. Sequences in development of the normal anus (modified after Tench).

that the muscles of the perineum arise from the primitive musculus sphincter cloacae which is apparent in the 22 millimeter embryo. This undifferentiated muscle sheet soon divides into the sphincter ani externus and a musculus sphincter sinus urogenitalis, attendant upon the division of the cloaca into the anal and urogenital tracts. The sphincter sinus urogenitalis subsequently further divides into the various muscles of the urogenital portion of the perineum.

The central depression of the anal mound will have a floor, and thus be separated from the hindgut, only in the rare instance of persistence of that dorsal portion of the cloacal membrane known as the anal plate. As has

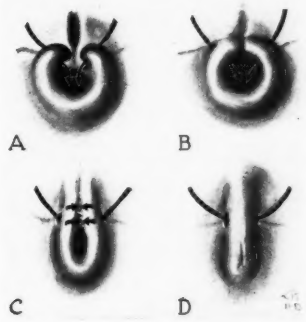

Fig. 5. Conjectural explanation of the manner of formation of the median sagittal band with bilateral fistulas.

Fig. 6. Photograph of the anus of a 1 month old baby. A small bit of mucus can be observed coming from an aperture anterior to the normal position for the anus. The two prominent folds of tissue are possibly remnants of the anal tubercles as represented in Figure 4 B and C.

previously been pointed out, however, this anal membrane is ordinarily already disappearing by the time of the formation of the anal mound. With the continued growth of the surrounding portions of the perineum the anal mound becomes relatively less elevated and finally disappears.

The anterior wall is actually formed by a three-way union. The two anal tubercles, approaching one another, are in turn being approached on their deep (cranial surfaces) by the down-growing urorectal septum. Not only should the anal tubercles fuse together in the midline, but their deep surfaces should meet and fuse with the lower edge of the urorectal septum and with the adjacent margins of the external cloaca, which have just previously approached each other and fused in the midline in coalition with the down-growing urorectal septum, thus completing the separation between the rectal and urogenital tracts.

TABLE I.—SUMMARY OF 6 CASES OF CONGENITAL MEDIAN BAND OF ANUS

Case No.	Associated pathologic conditions	Treatment	Result
1	Stenosis of anus	Excision of band and dilatation	Excellent
2	Stenosis of anus	Colostomy (?unnecessary)	Excellent
3	Stenosis of anus	Dilatation	Excellent
4	Stenosis of anus	Excision of band and dilatation	Excellent
5	Imperforate anus with rectoperineal fistula	Excision of band anoplasty	Excellent
6	Stenosis of anus with rectourethral fistula	Colostomy (?unnecessary); plastic correction of stenosis; division of rectourethral fistula	Not known

The sequence of events whereby the anal tubercles, urorectal septum, and adjacent parts of the margins of the external cloaca all converge upon one another for a final fusion to form the perineal body, or the central perineal area, is of utmost importance. Delay of one part to arrive here at the proper time, or an inadequate degree of final fusion, becomes the simplest and seemingly most likely explanation of the several types of anomalies or malformations that may develop in the anal area.

To explain the anomalous cases reported in this paper, the following conjectural explanation is presented. If the anal tubercles, as they are approaching one another, should not concurrently become attached to the lower edge of the urorectal septum and adjacent margins of the external cloaca they would tend to overhang these latter structures and thus leave a crevice (occupied by arrows bilaterally in Fig. 5A). Subsequent fusion in the midline of the anal tubercles and the lower edge of the urorectal septum without the total obliteration of the previously described crevice could result in the persistence of two channels or fistulas (as indicated by arrows in Fig. 5B). If, now, the process of fusion of the anal tubercles were carried to excess, and continued dorsalward along the opposing or adjacent margins of the anal orifice, the main outlet could become obliterated, leaving only the 2 lateral fistulas with an elevated ridge of tissue present in the median plane (the median sagittal band of tissue in the authors' cases). This is portrayed in Figures 5C and D.

If the anal tubercles meet and unite with one another but fail to be properly fused to the urorectal septum and the adjacent right and left margins of the cloaca, it will be seen that a median perineal fistula placed between the scrotum and the anus could result. If anomalous fusion of the two borders of the anal aperture along the midline should progress to a point short of completely sealing the anal orifice, one could expect the retention of some

slight degree of an anal aperture passing through the median band. Partial, but inadequate, adherence of the anal tubercle to the overlying urorectal septum and adjacent margin of the cloaca might result in the persistence of a laterally placed dimple (Fig. 4B) whose floor was closed over by a thin membrane.

It is to be emphasized that the foregoing explanation of the mechanism of development of such anomalies is hypothetical and that no series of embryonic specimens are on record which substantiate these conjectures. Interestingly enough, we have recently seen an infant 1 month of age with an anomaly which is illustrated in Figure 6. A small bit of mucus can be observed coming from an aperture anterior to the normal position for the anus. The two prominent folds of tissue are possibly remnants of the anal tubercle as represented in Figure 4B and C.

We believe that the conformation of the anal structures in this case tends to confirm at least some of our conjectures.

TREATMENT

Table I shows a summary of the treatment of these cases. In 3 cases local excision and anal dilatation were sufficient. In 1 case excision of the band of tissue together with anoplasty was done. In 1 case division of a rectourethral fistula was necessary. In 2 cases in which a temporary colostomy was performed we believe that, with our present knowledge, simple excision and dilatation would have been sufficient.

All patients, we believe, have normal function although it is too early to be certain about Case 6.

SUMMARY

Six cases of congenital median band of the anus are presented. All are in male infants. In 5 of the 6 there has been an associated stenosis of the anus. In 2 of the cases there were other anomalies of the rectum.

An outline of the development of the anal region is presented together with a conjectural explanation of the events leading up to the development of the anomalies we have observed.

We believe that treatment should consist of simple excision of the band of tissue, together with dilatation of the anus, and a search for other abnormalities.

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Fig. 6. Photograph of the anus of a 1 month old baby. A small bit of mucus can be observed coming from an aperture anterior to the normal position for the anus. The two prominent folds of tissue are possibly remnants of the anal tubercles as represented in Figure 4 B and C.

previously been pointed out, however, this anal membrane is ordinarily already disappearing by the time of the formation of the anal mound. With the continued growth of the surrounding portions of the perineum the anal mound becomes relatively less elevated and finally disappears.

The anterior wall is actually formed by a three-way union. The two anal tubercles, approaching one another, are in turn being approached on their deep (cranial surfaces) by the down-growing urorectal septum. Not only should the anal tubercles fuse together in the midline, but their deep surfaces should meet and fuse with the lower edge of the urorectal septum and with the adjacent margins of the external cloaca, which have just previously approached each other and fused in the midline in coalition with the down-growing urorectal septum, thus completing the separation between the rectal and urogenital tracts.

TABLE I.—SUMMARY OF 6 CASES OF CONGENITAL MEDIAN BAND OF ANUS

Case No.	Associated pathologic conditions	Treatment	Result
1	Stenosis of anus	Excision of band and dilatation	Excellent
2	Stenosis of anus	Colostomy (?unnecessary)	Excellent
3	Stenosis of anus	Dilatation	Excellent
4	Stenosis of anus	Excision of band and dilatation	Excellent
5	Imperforate anus with rectoperineal fistula	Excision of band anoplasty	Excellent
6	Stenosis of anus with rectourethral fistula	Colostomy (?unnecessary); plastic correction of stenosis; division of rectourethral fistula	Not known

The sequence of events whereby the anal tubercles, urorectal septum, and adjacent parts of the margins of the external cloaca all converge upon one another for a final fusion to form the perineal body, or the central perineal area, is of utmost importance. Delay of one part to arrive here at the proper time, or an inadequate degree of final fusion, becomes the simplest and seemingly most likely explanation of the several types of anomalies or malformations that may develop in the anal area.

To explain the anomalous cases reported in this paper, the following conjectural explanation is presented. If the anal tubercles, as they are approaching one another, should not concurrently become attached to the lower edge of the urorectal septum and adjacent margins of the external cloaca they would tend to overhang these latter structures and thus leave a crevice (occupied by arrows bilaterally in Fig. 5A). Subsequent fusion in the midline of the anal tubercles and the lower edge of the urorectal septum without the total obliteration of the previously described crevice could result in the persistence of two channels or fistulas (as indicated by arrows in Fig. 5B). If, now, the process of fusion of the anal tubercles were carried to excess, and continued dorsalward along the opposing or adjacent margins of the anal orifice, the main outlet could become obliterated, leaving only the 2 lateral fistulas with an elevated ridge of tissue present in the median plane (the median sagittal band of tissue in the authors' cases). This is portrayed in Figures 5C and D.

If the anal tubercles meet and unite with one another but fail to be properly fused to the urorectal septum and the adjacent right and left margins of the cloaca, it will be seen that a median perineal fistula placed between the scrotum and the anus could result. If anomalous fusion of the two borders of the anal aperture along the midline should progress to a point short of completely sealing the anal orifice, one could expect the retention of some

slight degree of an anal aperture passing through the median band. Partial, but inadequate, adherence of the anal tubercle to the overlying urorectal septum and adjacent margin of the cloaca might result in the persistence of a laterally placed dimple (Fig. 4B) whose floor was closed over by a thin membrane.

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STRUMA LYMPHOMATOSA

KEN W. BLAKE, M.D., F.A.C.S., Beverly Hills, California, and
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IN 1912, Hashimoto first reported upon 4 cases of a peculiar type of goiter. This he called struma lymphomatosa. It is surprising how clear were his clinical and pathologic observations. During the years intervening one phase of the histologic picture, namely, the marked lymphocytic infiltration of the gland, has stood out with such prominence that, with the exception of a few isolated instances, it has completely overshadowed and clouded the investigative horizon. Consequently there has been little attention directed to the epithelium, the colloid, the reticuloendothelial cells, or the vascular or neural structures. Recently some impetus has been given to the study of the epithelial changes, particularly by the work of Joll, Graham (8), and Parmely and Hellwig in their very fine histopathologic reports and to the study of the colloid and macrophages by Chesky, Dreese, and Hellwig. It is the purpose of this paper to focus still further attention upon the epithelial changes. The authors of this paper have studied 16 cases of struma lymphomatosa, or Hashimoto's disease, over the past 10 years. It has been possible to follow these cases carefully as they were all encountered in private practice. The summary of each of the 16 cases fits well into the clinical picture of this entity, for such Hashimoto's disease now seems to be.

The incidence of occurrence of the disease in this series among other surgical pathologic disorders of the gland is 3.3 per cent. This is somewhat higher than Marshall's 78 cases in 25,000 patients or, an incidence of .31 per cent from the Lahey Clinic in Boston. McSwain and Moore reported an incidence of .75 per cent from the New York Hospital. Joll of England reported an incidence of 0.9 per cent in 5,650 patients and Chesky, Dreese, and Hellwig of Halstead, Kansas, an incidence of 7.1 per cent in 2,031 patients. Whether this variation in geographic distribution is coincidental or actual is difficult to determine, although the span does appear to be great enough to lend some significance. Davison and Letton believe there has been a definite increase in the incidence of the disease since the advent of World War II.

In this series all patients have been of the female sex. There is only an occasional case in the male reported in the literature—a total of 9 apparently substantiated cases, to our knowledge.

The average age has been 47.8 years, the youngest being 19 and the oldest 70 years. The literature presents a span of the average age from 37.7 to 57.6 years.

The average duration for the known presence of the goiter is 4½ years, the shortest interval being 2 months and the longest 10 years.

The social status of the individual appeared to have no influence on the disease.

The most common presenting symptoms were those due to pressure on the trachea or esophagus and, possibly, the recurrent or superior laryngeal nerves. These were manifested by dyspnea, choking—particularly in the supine position with the neck extended—cough, dysphagia, and a husky voice. This latter condition was seen in 3 of the 16 patients. One or more of these pressure symptoms was seen in 75 per cent of the cases. There was no pain or tenderness in any of the cases here included. Those with tenderness may belong to the class of lymphoid thyroiditis of Crile (3), although as yet, it is not understood whether this is a distinct type of thyroiditis or just an early stage of struma lymphomatosa.

The other symptoms in the order of frequency were palpitation and tachycardia, fatigue, slightly increased nervousness, and a tendency to gain or maintain weight.

It is of interest that only 3 patients gave a history of normal menstrual periods at the time of surgery—6 being beyond the climac-

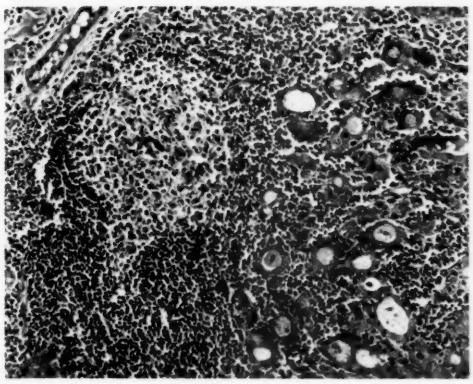

Fig. 1. Struma lymphomatosa showing lymphoid infiltration and the small thyroid follicles containing little colloid. ×95.

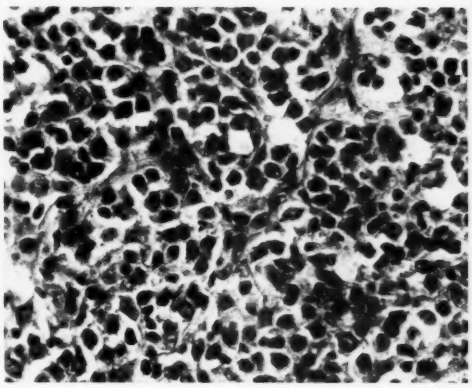

Fig. 2. Germinal center in lymph follicle with large and small lymphocytes, plasma cells, and reticulocytes. ×315.

teric, 4 having undergone hysterectomy, and 1 with bilateral oophorectomy 7 years previous to thyroidectomy. The 19 year old patient had 2 babies, 3½ and 15 months of age with no menses during the months intervening. Only 4 patients had taken estrogens. Parmely and Hellwig thought this altered ovarian function might bear some causal relationship to the disease.

The preoperative basal metabolic tests ranged from −32 to +12 per cent, the average being −8 per cent, a figure identical to that of Crile (2). A blood organic iodine determination was made on only 1 patient—this being 3.9 micrograms per cent. Statland, Wasserman, and Vickery report values of 2.5, 0.9, and 4.4 micrograms per cent in each of 3 patients. Thus, all 4 patients appear to be in the hypothyroid range. These authors performed radioactive iodine uptake studies on 7 cases and found the uptake varied from 15.6 to 58 per cent with an average of 33.6 per cent, which is in the lower range of normal.

Clinically, on palpation the goiters were all diffusely enlarged from 2 to 5 times normal size and all presented some prominence of the isthmus. The glands were rubbery to hard and, in several, note was made that the upper poles were blunt. There was no tenderness and no palpable thrill or audible bruit. There was no fixation to the prethyroid structures.

There were no obvious signs of hypothyroidism or hyperthyroidism. The pulse averaged 82 and all but 3 patients presented blood pressures within a normal range. These 3 had moderate hypertension.

Preoperative roentgenograms of the chest showed no substernal extension or mediastinal adenomas. The blood counts, serology, and urinalysis were all within the normal range. There was no evidence of a lymphocytosis.

Surgery was performed in each case in order to relieve pressure and to rule out a malignant lesion. Such surgery consisted of frozen section biopsy of both lobes of the gland followed by subtotal thyroidectomy, care being taken to clean all of the isthmus and pyramidal lobe from the trachea. As much of the lateral lobes as possible was left in situ and the amount removed varied from 11 to 136 grams. Total removal of a lobe was never deemed advisable or necessary. In 1 case a retrotracheal extension had to be removed. Exploration posteriorly is quite essential, especially in those patients with dysphagia or those with a husky voice, as this gland may grow to encircle the trachea completely. Silk or cotton has been used as ligature suture material and the wounds have been closed without drainage. The skin was reapproximated with clips that were removed in from 24 to 48 hours. There have been no deaths and no recurrent nerve injuries. Other methods of treatment available are x-radiation, desiccated thyroid orally, cortisone, or ACTH. X-ray therapy is advocated by some as being preferable to surgery

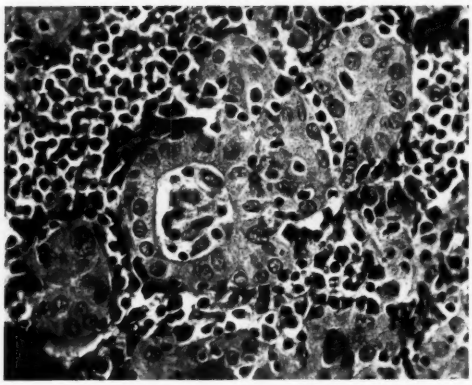

Fig. 3. Thyroid follicle with lymphocytes and epithelial cells in center and tall cuboid epithelium lining the follicle. Note "interfollicular cells" outside of the follicle. ×300.

and certainly can be resorted to in the poor risk patient. However, our first objection to this means of therapy is the inability to rule out malignant disease even though needle biopsies are taken. Our second objection is the fact that the level of postoperative hypothyroidism and myxedema cannot be as well gauged as with an adequate but not radical resection. Desiccated thyroid in moderately large doses will perhaps cause some remissions, particularly in the early cases. Cortisone and ACTH will cause a temporary decrease in the size of the gland but there is usually a recurrence of the goiter when therapy is discontinued. While antithyroid drugs, as thiouracil and its derivatives, have a specific and beneficial place in the treatment of subacute thyroiditis, they have no apparent place in the treatment of struma lymphomatosa. Radioactive iodine is usually taken up with insufficient concentration to allow for effective and accurately controlled dosage.

Postoperatively there has been only 1 recurrence. This recurrence was first noted 13 months after thyroidectomy in a 34 year old patient. The gland was treated 3 months later with x-radiation, 400 roentgens to each lobe, and receded in size to such an extent that it was no longer palpable. This resulted in marked symptoms of hypothyroidism with myxedema. Three months following completion of radiation the patient complained of increasing choking and dyspnea. Surgical exploration was reverted to and a dense band of scar tissue in the region of the removed isthmus was found pulled tightly across the trachea by the receding and contracting lateral lobes. Biopsy specimens were taken and the scar tissue was resected. In spite of the recent radiation the remaining glandular tissue appeared essentially unchanged from that obtained at the first operation 21 months previously. This second surgery was performed 3 years ago and the patient has since remained well with the exception of hypometabolism. Fifteen of the 16 patients have developed signs or symptoms of hypometabolism following surgery and the average basal metabolic rate has fallen from −8 to −18 per cent. Several, like those of Crile (2), have not shown too satisfactory a response to desiccated thyroid. Womack in 1944 pointed out that the changes noted in this disease were often seen in patients with adrenal cortical insufficiency. Jaffe, however, was of the opinion that they are separate although probably co-ordinate processes. Cortisone in doses up to 100 milligrams daily has been used in 2 of these patients for a period of 2 weeks with no apparent benefit.

Pathologically, there is no pathognomonic picture characteristic of struma lymphomatosa. Yet if one considers the gross and histologic features, together with the clinical features, the disease does seem to be a definite entity and certainly does not appear to be just a stage in the development of Riedel's struma as hypothecated by Ewing in 1922 and later refuted by Graham (7) in 1931.

The gland is diffusely and symmetrically enlarged, the isthmus and pyramidal lobe likewise sharing in this enlargement. As total thyroidectomy was not carried out in any of our cases, it was impossible to weigh the entire gland. However, they ranged from approximately 2 to 10 times the weight of the normal gland. The contour is usually regular —only 1 of our patients presenting any small nodules. The surface is slightly lobulated and the capsule may be somewhat thickened along the tracheal groove. The color may vary from a delicate pink, through a gray-white and pale yellow, to a very light tan. As the disease does

not extend beyond the capsule there are usually no adhesions to the overlying muscles and fascia. The cut surface is friable to rubbery in consistency and is likewise lobulated from an apparent increase in the interlobular connective tissue stroma. It is moist but relatively avascular and there is no colloid grossly visible. Suppuration and necrosis were never seen. In general, in the typical case, the surface is quite homogeneous throughout.

Microscopically there is everywhere a diffuse infiltration with lymphocytes as well as many focal areas of dense lymphoid tissue. This infiltration is within the stroma as well as in the interlobular and intralobular spaces. Within the focal aggregations one may see many distinct lymph follicles with a central germinal center. Within the centers there are both large and small lymphocytes, plasma cells, reticular cells, and occasionally an eosinophil. A scattering of lymphocytes may show mitosis. By far the majority of cells invading the stroma are small lymphocytes with a varying number of plasma cells, exhibiting their eccentric nuclei. In none of the above cases did we find giant cells as described by Graham (8) and by Womack. While this lymphoid invasion is very impressive we are in complete agreement with Parmely and Hellwig that this tissue does not compose more than one-third of the entire bulk of the gland.

The stroma throughout the gland is somewhat hyperplastic and there is also some slight increase in the intralobular areas between the follicles. The capsule is likewise somewhat thickened. However, in none of our sections does this fibrosis become so marked as to replace large portions of the gland completely, so commonly seen in Riedel's struma. The vessels show no changes other than those coincidental to the age of the patient.

The changes in the thyroid epithelium and the marked loss of colloid warrant further investigation, and evidence has been presented that these changes once established are rather stationary and permanent. This fact, as previously noted, we have been able to verify by the study of the tissues taken at an interval of approximately 21 months from our 1 patient with recurrence. Most of the sections show almost a complete loss of colloid—that remaining being mostly eosinophilic. Occasionally small droplets of this colloid appear to be lying entirely without the acinus, between clumps and strands of epithelial and phagocytic cells. However, we have not observed that these droplets are selectively surrounded by lymphocytes as reported by Chesky, Dreese, and Hellwig. Neither have we observed that they form the nucleus about which the lymph follicles with their germinal centers form. There are relatively few colloid vacuoles in our specimens. Chesky, Dreese, and Hellwig in quite recent work have noted under the electron microscope a similarity between the colloid in struma lymphomatosa and that of the rabbit thyroid stimulated by thyrotropic hormone, both appearing different from that of normal thyroids. The acinar lumen may contain free lymphocytic, phagocytic, and epithelial cells. The acinar epithelium ranges from regular low cuboid to an irregular high cuboid or columnar type. The acinus that retains any amount of colloid is usually lined with a low somewhat compressed cuboid cell, whereas those that contain only little colloid or cellular debris are usually lined with tall columnar cells. These cells are regularly arranged, even, and present smooth cell membranes. They present relatively large and well rounded nuclei either centrally or basally placed which contain usually 1 to 2 rather large nucleoli. Their cytoplasm may be homogeneous and smooth, although many will present granules. In these

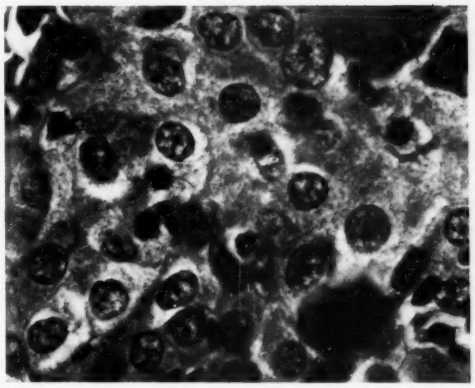

Fig. 4. The "interfollicular cells" and a droplet of "extrafollicular" colloid (lower right). ×630.

sections the majority stain definitely eosinophilic and appear active and regenerating although in some areas neutrophilic cytoplasm predominates. Dempsey, Dinsmore, and Hazard have attempted to correlate radioactive iodine avidity with these cells by use of radioautographs. They found no radioactivity in areas of acidophilic epithelial cells but a significant uptake in areas of neutrophilic cells. Statland, Wasserman, and Vickery, on the other hand, were unable to correlate radioactive uptake with the histologic picture in specific areas of the gland. In the acini that contain no colloid the cells become large and somewhat polygonal in shape with a lighter and more delicate pink staining of the cytoplasm which appears definitely granular and may be vacuolated. In these, the cell membranes are often indefinite. The nuclei also appear slightly larger or swollen and are still seen to contain 1 or 2 nucleoli. These have a fine chromatin network. Without doubt some of them appear to be degenerating, presenting ill-stained coarsely granular cytoplasm with many vacuoles and karyolytic nuclei that are mere shells with little chromatin. There are also many epithelial cells lying apparently outside of these regular follicular groups which are commonly called "interfollicular cells." Some believe they are of a different type but the authors believe they are mainly tangentially cut follicular walls. There has been much discussion as to whether the follicular cells are undergoing hyperplasia or degeneration. From our observations it appears to us that there is evidence of both hyperplasia and degeneration taking place simultaneously in the same gland, the hyperplasia, however, predominating. That many of these cells exhibit hypertrophy, we believe, cannot be denied and it may well be of a compensatory nature.

The etiology of the disease is still much a mystery, although many theories have been advanced, and whether or not these epithelial changes are the cause or the effect is still unknown. Unless one wishes to accept the mere presence of lymphocytes as denoting an inflammatory reaction there is little to substantiate the placement of this disease in the realm of a chronic thyroiditis and in our opinion, as Graham (7) has so ably stated, "the lesion may be considered primarily to be a local manifestation of a constitutional disorder, the nature of which is as yet not understood."

SUMMARY

Sixteen cases of struma lymphomatosa (Hashimoto's disease) are reported in summary.

The incidence of occurrence among other surgical diseases of the thyroid of 3.3 per cent is the first reported in the western coastal area. The cases fit well into the sex, age, clinical, and pathologic criteria for the disease.

Subtotal thyroidectomy has been selected as the method of choice for treatment as pressure symptoms are relieved, malignant disease is ruled out, and postoperative hypothyroidism is kept to a minimum.

Hypometabolism resulted in 15 of the 16 cases. Myxedema, however, only followed radiation in 1 patient with a recurrence of growth. Several of these have not shown too satisfactory a response to desiccated thyroid therapy.

The gross and microscopic pathologic features are described and illustrated and attention is focused upon the epithelial elements. Both epithelial hyperplasia and degeneration appear to be taking place simultaneously, the former predominating.

Doubt is expressed in classifying this as a true inflammatory reaction or thyroiditis.

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FURTHER EXPERIMENTAL STUDIES AND OBSERVATIONS ON URETERAL REFLUX AND URINARY DIVERSION

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PYELONEPHRITIS from ureteral reflux following ureterointestinal anastomosis has become such a problem that the old operation of implanting the ureters in a rectum defunctionalized by permanent colostomy has been revived by Boyce and Vest. This is an extremely radical procedure, and while it may prove to be the only answer to the problem of pyelonephritis caused by ureteral reflux, search should be continued for better methods of urinary diversion.

Two such methods which differ from the standard ureterointestinal anastomosis and have distinct possibilities are the creation of a substitute bladder (Gilchrist procedure) and the interposition of an antiperistaltic segment of small bowel between the ureters and the sigmoid.

In the Gilchrist procedure the terminal ileum, cecum, and ascending colon are isolated; bowel continuity being re-established by ileocolostomy. The terminal ileum is brought out to the skin of the abdomen to serve as the new urethra. The ureters are transplanted to the new bladder created by the cecum and ascending colon. The new bladder achieves a high degree of continence so that no collecting device for urine is necessary after several weeks. The patient catheterizes the new urethra often enough to prevent overflow leakage, usually every 4 to 8 hours. Continence is maintained by the competency of the ileocecal valve, antiperistalsis of the new urethra, sufficient size of the new bladder, the tightness of the abdominal wall about the opening of the new urethra, and the bringing out of the new urethra well superior on the abdomen.

The ureters empty into a reservoir which is relatively clean, and the mucosa of which is infection resistant. Ureteral reflux should be lessened to a marked degree by removing the intracolonic pressures, and, when occurring, will be much less hazardous due to the absence of fecal material from the urinary reservoir. *Blood chemistry changes are no different from those of ureterosigmoidostomy.*

It is true that this is radical surgery, but in competent hands the mortality should not be high. The end result is certainly far more desirable than a permanent colostomy with ureterorectal anastomosis or death from chronic pyelonephritis following ureterosigmoid anastomosis. The advantages of urinary diversion by this method are: (1) no collecting device is necessary, (2) there is no soiling such as occurs with a wet colostomy, (3) the ureters are not contaminated by the fecal stream, (4) cystoscopy of the substitute bladder is possible, and (5) there is no limitation of radical operations when dealing with pelvic cancers.

The Gilchrist operation must be done by many surgeons the country over, before it can be assessed and evaluated properly. Patients subjected to this procedure must be followed

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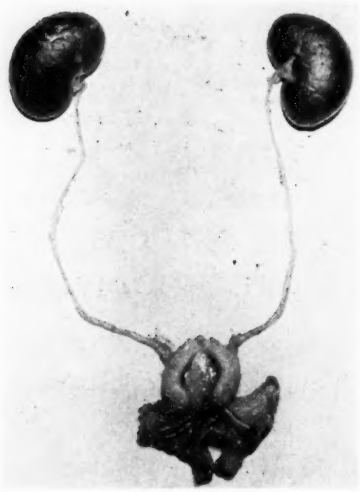

Fig. 1.

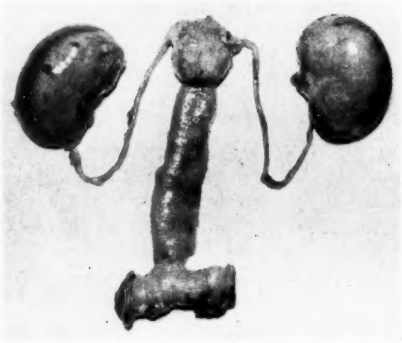

Fig. 2.

Fig. 1. Necropsy specimen of dog having vesicosigmoidostomy.
Fig. 2. Necropsy specimen of dog having antiperistaltic ileal loop anastomosed between urinary bladder and sigmoid.

for years with pyelograms, kidney function studies, and blood chemistry determinations. This procedure appears to me, at the present time, to be the most practical form of urinary diversion which offers the patient comfort plus freedom from renal failure resulting from chronic pyelonephritis in turn caused by ureteral reflux of fecal material from the colon.

The interposition of an antiperistaltic segment of small bowel between ureters and sigmoid was first reported by Berg in 1907 and has certain limitations. It is fairly certain that ureteral reflux of colonic material would not be entirely prevented because the length of the antiperistaltic loop is limited by the degree of absorption of urine. Also this method requires preservation of the rectum as a urinary reservoir thus limiting its value in radical pelvic surgery. Cystoscopic observation of the ureteral orifices, of course, would be impossible. For clarity I would like to quote the summary of experimental work concerning this method of urinary diversion by Rieger and Harris which was published previously.

"A group of dogs was operated upon in an attempt to find a method of reducing the hazards and occurrence of ureteral reflux following ureterointestinal anastomosis. This was done by interposing an antiperistaltic segment of small bowel between the urinary tract and sigmoid colon in order that peristalsis would protect the urinary tract from contamination by colonic contents. Clinically, the ureters would be transplanted to the proximal end of this isolated loop and the sigmoid colon anastomosed to the distal end. Experimentally, the dome of the urinary bladder was anastomosed to the proximal end of the isolated loop, and the urethra was ligated so that end results would not be confused by difficulties with ureteral transplants. Continuity of the bowel was re-established by ileoileostomy. Ileal loops of 15 to 20 centimeters were compatible with life in regard to absorption of urine at least up to 4 months. Ileal loops 35 centimers or more in length absorbed so much urine that they were incompatible with life. Even these long loops were not of sufficient length to prevent reflux contamination of the urinary bladder by colonic contents. A corollary may be drawn that infectious material cannot be entirely prohibited from ureters transplanted to similar ileal loops, though probably the degree of ureteral reflux with resulting infection can be reduced. More experimental work on the problem is necessary."

It is this further experimental work that I now wish to report.

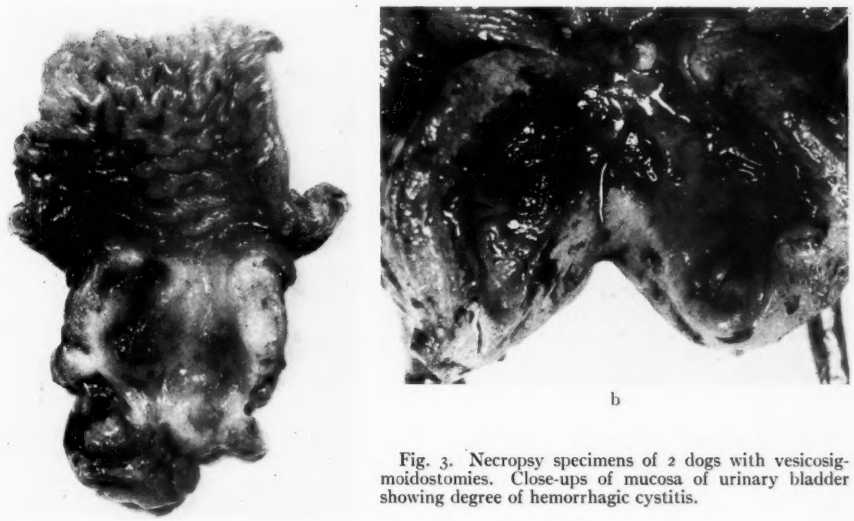

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Fig. 3. Necropsy specimens of 2 dogs with vesicosigmoidostomies. Close-ups of mucosa of urinary bladder showing degree of hemorrhagic cystitis.

METHODS AND RESULTS

Five dogs were operated upon under intravenous nembutal anesthesia. The dome of the urinary bladder was anastomosed directly to the sigmoid colon, after which the urethra was divided and ligated. All urine thus passed from the ureters into the urinary bladder and thence to the sigmoid colon. These dogs were followed with blood chemistry determinations of nonprotein nitrogen, carbon dioxide combining power, chlorides, and potassium (Figs. 1 and 2).

Four other dogs were operated upon with the interposition of an ileal loop 15 to 20 centimeters in length between urinary bladder and sigmoid colon as in our previous work. This second group was for comparison use.

Barium enema studies were done upon all dogs to compare the ease with which the urinary bladders could be filled in the two groups. No detailed pressure studies were done, but it was obvious that it was much more difficult to fill the bladders with the interposed ileal loop than those without the interposed loop.

As would be expected the blood chemistry determinations in the first group followed a pattern similar to that following bilateral ureterosigmoidostomy; that is, the nonprotein nitrogen rose moderately and then gradually fell over a period of months, never reaching normal, the carbon dioxide combining power fell moderately to level off at about 40 volumes per cent, the chlorides rose very slightly, and the potassium remained essentially unchanged but with fairly wide variations within normal values. These changes were similar in both groups, although a little more extreme in the group with the interposed ileal loop (see Fig. 2 in ref. 5, p. 95).

All dogs were sacrificed at 4 months by electrocution and careful autopsies were done on all. The degree of gross urinary bladder infection in each group was compared. A severe hemorrhagic cystitis was present in 4 of the cases in the vesicosigmoidostomy group (Fig. 3). In the group with an interposed ileal loop slight to moderate cystitis was apparent (Fig. 4).

DISCUSSION

The question that I am trying to answer is this. Can an antiperistaltic ileal loop anastomosed between ureters and sigmoid in clinical urinary diversion be expected to reduce or prevent infection of the urinary tract caused by reflux of colonic contents as compared to the infection following direct ureterosigmoidostomy?

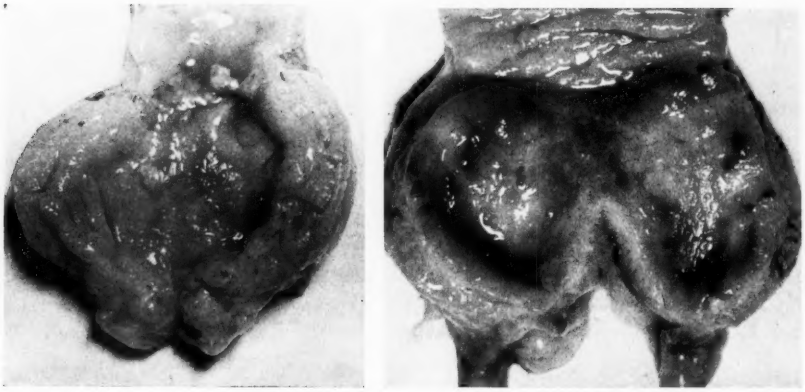

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Fig. 4. Necropsy specimens of 2 dogs having an antiperistaltic ileal loop anastomosed between urinary bladder and sigmoid. Gross cystitis is present, but not to the degree seen in Figure 3.

Previous experimental work has established that an antiperistaltic ileal loop 15 to 20 centimeters in length anastomosed between urinary bladder and sigmoid was compatible with life in regard to urinary absorption, at least up to 4 months. The dome of the urinary bladder was anastomosed to the proximal end of the ileal loop and the urethra was ligated in order to avoid the well known difficulty with ureteral transplantation in dogs. Ileal loops 15 to 20 centimeters in length were compatible with life but did not prevent infection of the urinary bladders. Blood chemistry changes were similar to, though slightly more extreme than, those following ureterosigmoidostomy.

The present experimental work demonstrates that infection in a urinary bladder protected by an antiperistaltic ileal loop is much less than that in a bladder anastomosed directly to the sigmoid. Barium enema studies indicate that it is much more difficult to manipulate the barium into a urinary bladder separated from the colon by an antiperistaltic ileal loop than into a bladder anastomosed directly to the sigmoid. Blood chemistry studies show that the interposed ileal loop of 15 to 20 centimeters in length causes a slight increase in the known variation following ureterosigmoidostomy; that is, the nonprotein nitrogen is slightly more elevated, the carbon dioxide combining power is slightly more depressed, and the chlorides slightly, if at all, more elevated. The serum potassium apparently is not markedly affected and showed an irregular variation. In humans, a high fluid intake, frequent evacuation of the rectum, and oral sodium bicarbonate or sodium citrate should aid in reducing the acidosis due to hyperchloremia. Obviously it is very difficult to attempt this experimentally in dogs.

These studies indicate that in clinical cases of urinary diversion by ureteral transplantation, an antiperistaltic ileal loop interposed between ureters and sigmoid could be expected to reduce considerably the urinary tract infection caused by reflux of colonic contents. If this proved to be the case, deaths from chronic pyelonephritis following ureterointestinal anastomosis would be reduced or postponed.

Of course, acidosis from urinary absorption would be more of a problem than in ureterosigmoidostomy. However, as Lapides has recently pointed out, many cases of acidosis following ureterosigmoidostomy are not due purely to absorption of urine from the colon but are partly due to the inability of kidneys, damaged by infection or obstruction, to handle the absorbed urine. Reduce this infection by reducing ureteral reflux and reduce obstruction by performing mucosa-to-mucosa anastomoses and the cases of acidosis from urinary absorption may also be reduced.

Urine apparently has no more injurious effect on ileum than it does on any part of the colon. This had been personally observed before in the experimental development of the Gilchrist or substitute bladder operation.

The disadvantages of the interposed loop operation as compared to the Gilchrist procedure are that urinary absorption would be more of a problem and that ureteral reflux of infected colonic contents could not be entirely prevented. The one outstanding advantage, of course, would be the retention of a urinary reservoir with an outlet controlled by a voluntary muscle, the anal sphincter.

The very distinct advantage, in regard to ureteral reflux, that the Gilchrist procedure has over the interposed ileal loop operation is that intraluminal bowel pressure transmitted directly up the ureters is completely eliminated. Some reflux undoubtedly would occur at times such as when straining at stool or coughing. However, this reflux should be relatively harmless, as the intestinal mucosa of the substitute bladder is infection resistant, the only thing in the urinary reservoir available for reflux is urine, and this urine can be rendered sterile, or almost so, if aseptic technique is used in the self-catheterization.

Both procedures deserve a clinical trial in large centers with many candidates for urinary diversion. The operative mortality should not be prohibitive in competent hands, and, I believe, should ultimately be less than ordinary ureterosigmoidostomy when late deaths due to pyelonephritis caused by ureteral reflux are taken into account.

SUMMARY

A group of dogs with direct vesicosigmoidostomies has been compared with another group of dogs having an antiperistaltic ileal loop anastomosed between bladder and sigmoid as to degree of urinary bladder infection, ease of regurgitation of contrast media from sigmoid to urinary bladder, and blood chemistries. In the second group the urethras were ligated and bowel continuity was re-established by ileoileostomy. In the group with direct vesicosigmoidostomies infection of the urinary bladder was more severe, it was far easier to cause barium to pass from the sigmoid into the urinary bladder and blood chemistries were similar to, but not so extreme as, the second group.

Previous experimental work had established that an antiperistaltic ileal loop 15 to 20 centimeters in length anastomosed between urinary bladder and sigmoid with the urethra ligated was compatible with life in regard to urinary absorption from bowel.

The corollary may now be drawn that in clinical cases of urinary diversion ureteral reflux with resulting pyelonephritis following ureterointestinal anastomosis should be decreased by the interposition of an antiperistaltic ileal loop between ureters and sigmoid. The procedure awaits further investigation and clinical trial.

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A MORPHOLOGIC STUDY OF THE MYENTERIC PLEXUS AND MUSCULATURE OF THE PYLORUS WITH SPECIAL REFERENCE TO THE CHANGES IN HYPERTROPHIC PYLORIC STENOSIS

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NUMEROUS etiologic factors have been suggested for the development of congenital hypertrophic pyloric stenosis since Hirschsprung presented 3 cases of this condition in 1888. In order to understand the various etiologic theories, it is necessary to review briefly the neuromuscular anatomy and physiology of the pylorus.

McCrea, from dissections of the abdominal distribution of the vagus nerves, found that vagal distribution to the stomach was constant and neither the anterior or posterior vagus nerves sent branches directly to the stomach below the incisura on the lesser curvature. He found in all cases, however, that there was vagal innervation of the pylorus and that it came indirectly from the anterior vagus via the hepatic branches. These branches entered the pylorus along the course of the gastroduodenal artery. The posterior vagus nerve below the esophageal plexus did not seem to supply any fibers to the stomach below the angle on the lesser curvature. In contrast McCrea found that sympathetic fibers from the celiac plexus supplied all areas of the stomach.

Hill found that all vagal fibers (preganglionic) entering the wall of the stomach and intestine immediately formed synaptic relations with the associative or Dogiel type I cells of the myenteric plexus lying between the longitudinal and circular muscles. These associative neurons in turn formed synaptic relations with motor or Dogiel type II cells which sent nerve fibers directly to individual muscle fibers in the longitudinal and circular muscle layers. Hill was unable to find any synaptic relations in the myenteric plexus with sympathetic postganglionic fibers, and these fibers were invariably traced directly to the submucosa where fibers terminated directly in submucosal glands and in mucosa.

At the upper end of the pylorus M'Crea, M'Swiney, Morison, and Stopford described a transverse band or the lower segment sling of Forssell dividing the pylorus into a functional unit from the cardia. Cathcart described a prepyloric sphincter at the junction of the cardia and pyloric antrum. Horton found a constant block of fibrous tissue separating the circular muscle of the pylorus from that of the duodenum in all specimens dissected. This block did not occur in the longitudinal muscle.

Three types of gastric waves have been demonstrated by the photokymographic balloon according to Hightower. The type I or tonus wave is seen throughout the stomach. The type II or peristaltic wave has two phases according to May, Klein, and McCrea. One phase which is arrhythmic and of low amplitude originates in the cardia and passes on the lesser curvature only to the junction of the cardia and the pylorus. At this point an entirely new wave of high amplitude is initiated every 15 to 18 seconds. This wave is rhythmic and passes directly to the pyloric sphincter. The point of initiation of this antral wave on the lesser curvature has been called the "pacemaker" by Alvarez and Hosoi and has been demonstrated in excised strips of muscle. The third wave is a summation wave of types I and II.

M'Crea and M'Swiney have demonstrated in dogs that stimulation of the vagus nerves

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produced 89 per cent motor and 11 per cent inhibitory responses, and that stimulation of splanchnic nerves produced 63 per cent motor and 37 per cent inhibitory responses in the pylorus (22, 23). Thomas and Wheelon found that the response of the pylorus depended on its state of activity at the moment. The fact that motor activity of the gastrointestinal tract originates in the myenteric plexus was demonstrated by Bayliss and Starling in the denervated gastrointestinal tract when motor activity was actually increased in cocainized segments. Hightower found that within 10 days after vagotomy the type II or peristaltic waves were entirely abolished from the human stomach. Then if 10 milligrams of urecholine were given subcutaneously, these waves not only returned temporarily but actually were increased in amplitude over the normal. The motor activity of the splanchnic nerves has never been explained anatomically.

The pyloric sphincter is normally closed according to the majority of investigators and only opens when a wave of gastric contents approaches. This mechanism was explained anatomically by Horton who demonstrated that in the region of the pyloric sphincter longitudinal muscle fibers pass through the circular muscle and insert into the submucosa and thus act as a dilator of the sphincter. Other factors governing the rate of gastric emptying are the volume of gastric and duodenal contents, the vagal and splanchnic stimulation, and the character of the food in the stomach and duodenum.

Thus it may be seen that any abnormal neurogenic impulse, particularly vagal, could produce severe abnormalities in function of the pylorus and pyloric sphincter.

The work hypertrophy and neurogenic imbalance theories of the cause of congenital hypertrophic pyloric stenosis are the most universally accepted. Tumpeer and Bernstein have shown that no obstruction occurred after a tumor similar to that in hypertrophic pyloric stenosis was produced in the pyloruses of dogs with liquid paraffin. By this means they indicated that primary hypertrophy of the muscle was not the etiologic factor. Thomson, after reviewing 100 cases of congenital hypertrophic pyloric stenosis, advanced the opinion that the pyloric hypertrophy developed in utero as a result of repeated gastric contractions on small quantities of amniotic fluid against a pylorus which failed to open. Lehmann stated that the pylorus is normally closed and thus any neuromuscular imbalance can cause work hypertrophy. On the other hand, Walton stated that congenital hypertrophic pyloric stenosis is not the result of spasm alone since atropine does not relieve the condition; there is no improvement with the stomach at rest, and the condition persists after death.

Herbst in 3 cases of congenital hypertrophic pyloric stenosis found evidence of disintegrating nucleoli and a loss of normal chromatin patterns in the myenteric ganglion cells of the pylorus. Stöhr found in the cytoplasm of these cells a loss of fibrillar pattern and vacuolization. Ingersoll produced identical myenteric ganglion changes in the cells of the rat after prolonged vagal stimulation.

As further evidence of the vagotonic state in hypertrophic stenosis of the pylorus, Bendix and Necheles followed infants with congenital hypertrophic pyloric stenosis into adulthood and found that these individuals had a much higher incidence of peptic ulceration and other gastrointestinal disorders than the rest of the population.

With this evidence of a possible vagotonic state leading to neurogenic imbalance and thus to congenital hypertrophic pyloric stenosis, the following study was undertaken to determine what if any neurologic changes could be found exclusively in the pyloric segment involved. Our plan of approach was to investigate the myenteric ganglion, nerve fiber tracts, and musculature in normal stomachs and duodenums of infants and compare conditions with those found in cases of hypertrophic pyloric stenosis.

MATERIALS FOR STUDY

Approximately 4,000 longitudinal and cross serial sections were examined under the microscope. These sections were cut from blocks of tissue taken from the wall of the stomach just below the incisura, in the midportion of the pyloric antrum and at the pyloroduodenal junction. The tissue studied

TABLE I.—AGE AND SEX OF PATIENTS AND FINDINGS AT OPERATION OR CAUSE OF DEATH

	Case	Sex	Age	Cause of death or findings at operation
Infants Normal stomachs	1	M	3 yr.	Burn; shock
	2	F	1.5 yr.	Traumatic shock
	3	M	5 yr.	Astrocytoma
	4	F	3 da.	Tracheoesophageal fistula; prematurity
	5	M	2 da.	Erythroblastosis
	6	M	2 da.	Prematurity
	7	F	Stillborn	Hydrocephalus
	8	F	5 mo.	Erythroblastosis
	9	M	Stillborn	Intrauterine asphyxia
	10	M	1.5 mo.	Congenital heart disease
Hypertrophic pyloric stenosis	11	F	4 mo.	Congenital heart disease
	12	F	3 wk.	Postoperative hemorrhage
	13	M	3 yr.	Tumor persisted; intestinal obstruction
	14	F	1 mo.	Pneumonia; amyotonia congenita
	15	F	2 mo.	Inanition; patent ductus arteriosis
	16	F	3 mo.	Ramstedt operation 1 mo. before; tracheal obstruction
	17	M	2 mo.	Repair of tracheoesophageal fistula
	18	M	14 da.	Inanition; subarachnoid hemorrhage
	19	M	25 da.	Repair of tracheoesophageal fistula
Adults Normal stomachs	20	M	23 yr.	Posttraumatic cerebral edema
	21	F	30 yr.	Drowning
	22	F	22 yr.	Acute hemorrhagic pancreatitis
	23	F	21 yr.	Acute leukemia
	24	F	27 yr.	Thrombocytopenic purpura
Persisting hypertrophic pyloric stenosis	25	F	18 yr.	Gastroenterostomy when 28 da. old; pyloric tumor persists
	26	M	24 yr.	Gastroenterostomy when 6 wk. old; pyloric tumor persists
	27	M	24 yr.	Gastroenterostomy when 5 wk. old; pyloric tumor persists
	28	M	22 yr.	Gastroenterostomy when 8 wk. old; pyloric tumor persists
	29	M	29 yr.	Gastroenterostomy when 5 wk. old; pyloric tumor persists

was obtained at necropsy or at operation. Tissue of four types was studied (Table I): (1) Specimens came from normal stomachs of 10 infants and children ranging in age from birth to 5 years. (2) Specimens in this group came from 8 infants and 1 child who had hypertrophic pyloric stenosis. The ages of the 8 infants ranged from 14 days to 4 months. The child, who was 3 years old, had died of intestinal obstruction, but the hypertrophic pyloric stenosis treated medically in infancy was still present. (3) Specimens came from normal stomachs of 5 adults ranging in age from 21 to 30 years. (4) Specimens were obtained at operation from 5 patients who had undergone gastroenterostomy in infancy for hypertrophic pyloric stenosis, and further operative procedures consisting of gastrectomy of the Billroth I type were performed at ages ranging from 18 to 29 years because of gastrojejunal ulcer or gastrojejunitis. All of these specimens in group 4 showed persistence of the hypertrophic pyloric stenosis. Specimens of adult stomachs removed by gastric resection after previous gastroenterostomy were not used as controls for comparison with group 4 even though no hypertrophic pyloric stenosis was present because the available specimens were complicated by active or inactive duodenal ulcer and, therefore, the condition of the pylorus could not be considered normal. The age and sex of the patients as well as the cause of death or findings at operation are given in Table I.

Little investigative work has been reported in the literature on the normal anatomy of the myenteric plexus and musculature of the stomach and duodenum, and thus it was necessary to study the normal stomachs of infants and adults to establish a basis for comparison with stomachs affected by hypertrophic pyloric stenosis.

METHOD OF CUTTING AND STAINING SERIAL SECTIONS AND OF REPORTING RESULTS

The average thickness of the microscopic sections cut was 0.008 millimeter, and since the width of the ganglia of the myenteric plexus is from 0.020 to 0.030 millimeter serial sections were necessary to study all aspects of these cells.

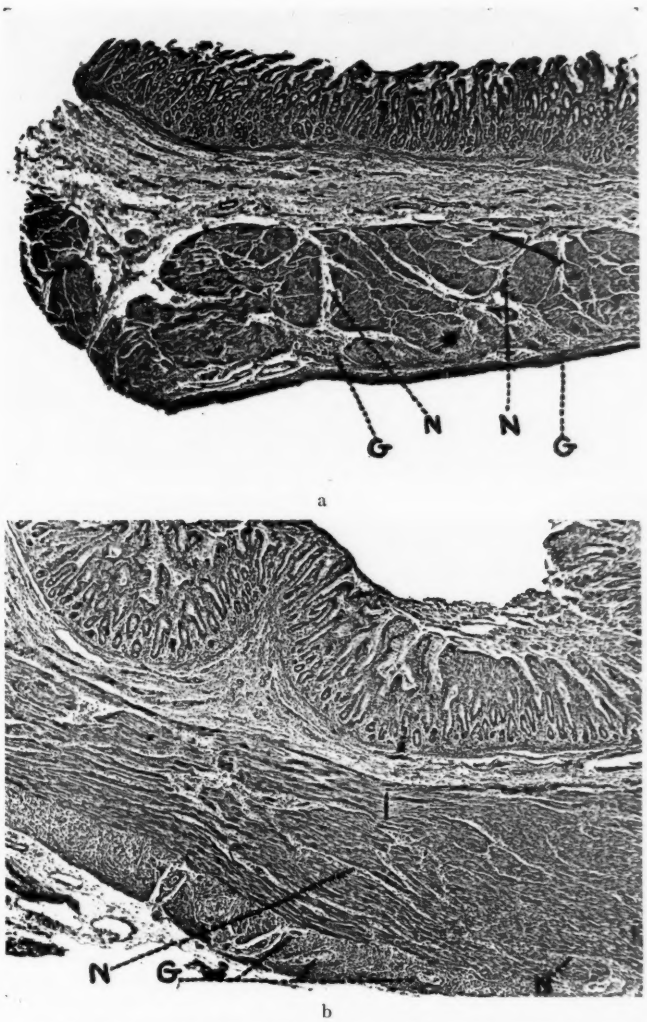


Fig. 1. Relationship of myenteric ganglion cells, nerve bundles and muscle thickness. a, In the normal infant stomach just below the incisura (cresyl violet, ×35). b, In a similar location in an infant with hypertrophic pyloric stenosis demonstrating little change from the normal (cresyl violet, ×35).
In all figures *G* stands for ganglion cells and *N* for nerve bundles.

Because of the size of the ganglion cells every third section was stained with cresyl violet for their best visualization. The other two sections consecutively were stained alternately with hematoxylin and eosin for visualization of muscle cells and by Bielschowsky's silver impregnation method (described by Mallory) for visualization of axons, dendrites, and nerve fibers. As is well known many difficulties occur in the silver staining of nerve filaments described by Kernohan, and, because no constant period of fixation in formalin was

possible before staining, any attempt to duplicate the work of Hill in tracing the course of axons and dendrites was abandoned. The silver-stained preparations in this study were used merely for quantitative estimation of the numbers of nerve fiber bundles.

In screening the suitability of the various blocks of tissue for study, 20 serial sections were cut and stained as described from each end of the block, and, if the microscopic findings in these sections proved significant, further sections were taken from the same block. An average of 6 blocks of tissue was removed from each specimen, 2 each from the stomach, pylorus, and junction of the pylorus and duodenum.

The numbers of ganglion cells and nerve tracts seen in these sections were graded quantitatively on the basis of 1 to 4; 1 signifies only occasional; 2, few; 3, frequent; and 4, numerous cells or tracts. This grading was applied to the number of cells and nerve fibers as judged from observations of all serial sections in the same area, and Figures 1 to 4 show the average in each of these areas.

Likewise, muscular thickness was graded 1 to 4 in the various areas; 1 represents normal thickness of the infant duodenum; 2, twice the thickness of 1; 3, three times the thickness of 1; and 4, four or more times the thickness of 1. All of these observations were made by one person. This method prevents any error due to uncorrelated observations. The grades were averaged to show trends as objectively as possible.

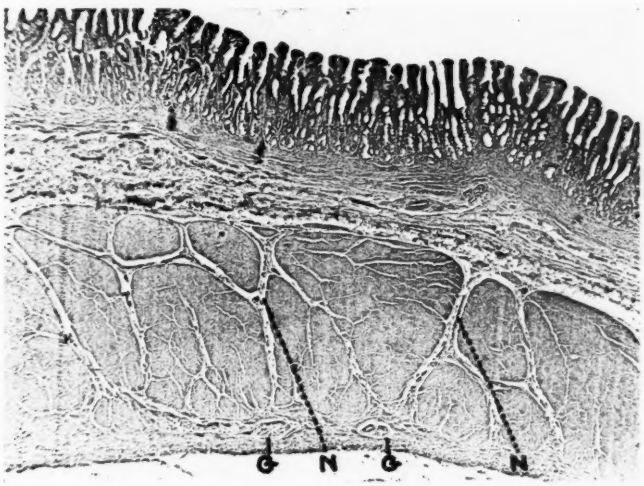
THE NUMBER OF GANGLION CELLS, NERVE FIBER TRACTS, AND MUSCLE THICKNESS IN VARIOUS SEGMENTS OF STOMACH, PYLORUS, AND DUODENUM

The majority of the myenteric ganglia were found in a plane between the longitudinal and circular muscle layers of the stomach and duodenum. This observation confirms the findings of Kuntz. Ganglia were found occasionally along the course of the nerve tracts running through the circular muscle to the submucosa, but no ganglion cells were seen in the submucosa.

In the normal infant and adult the number of ganglion cells and nerve tracts increased from the incisura of the stomach down to the termination of the pylorus as did the thickness of the musculature. At the onset of the duodenum, however, the number of ganglion cells and nerve tracts and the thickness of the muscle decreased abruptly to less than in the stomach below the incisura (Table II and Figs. 1 to 4a).

However, in the presence of hypertrophic pyloric stenosis of infants as well as of adults we found an apparently definite decrease from the normal in the number of ganglion cells and nerve tracts accompanied by a definite increase in thickness of the circular muscle (grade 4+) in all the pyloric segments studied. This decrease in myenteric ganglion cells is real rather than apparent because the circumferential plane in which they lie in the pylorus is not increased very much in hypertrophic pyloric stenosis, but rather, the lumen of the pyloric antrum is decreased. Little change from the normal had occurred in the number of ganglion cells and nerve tracts and thickness of muscle of the stomach above the pylorus. There was a slight increase in the number of ganglion cells and nerve fiber tracts in the duodenum, but no noticeable change in the duodenal musculature.

It is, of course, realized that such methods of estimating the numbers of ganglion cells and nerve bundles and measuring muscle thickness give only approximations and do not allow for the fact that, as circular muscle hypertrophies, the ganglion cells will be spread further apart. However, the diminution in numbers of ganglion cells and nerve fiber bundles in proportion to circular muscle thickness seems to occur because there is actually little gross increase in the diameter of the pylorus in pyloric stenosis, but rather the increase in thickness of the circular muscle causes an encroachment on the submucosa and pyloric lumen and does not cause marked stretching of the visceral peritoneum. Therefore, there is no marked increase in perimeter of the plane in which the myenteric ganglia lie. Thus, since it was impossible accurately to enumerate and measure these relationships from our method of study and since our results are only approximate, no definite conclusion can be made as to the



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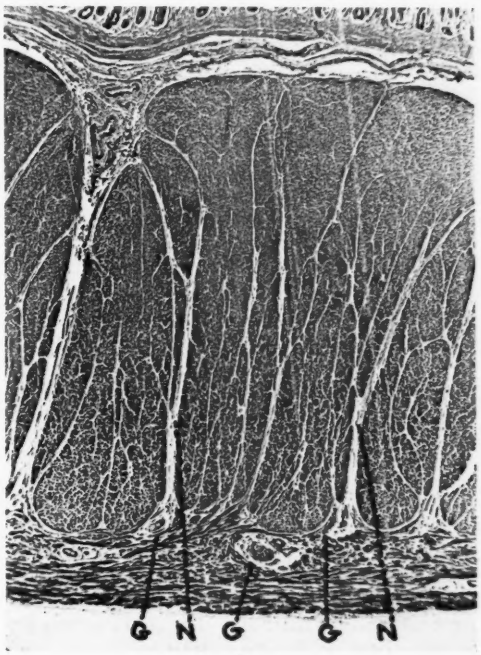


b

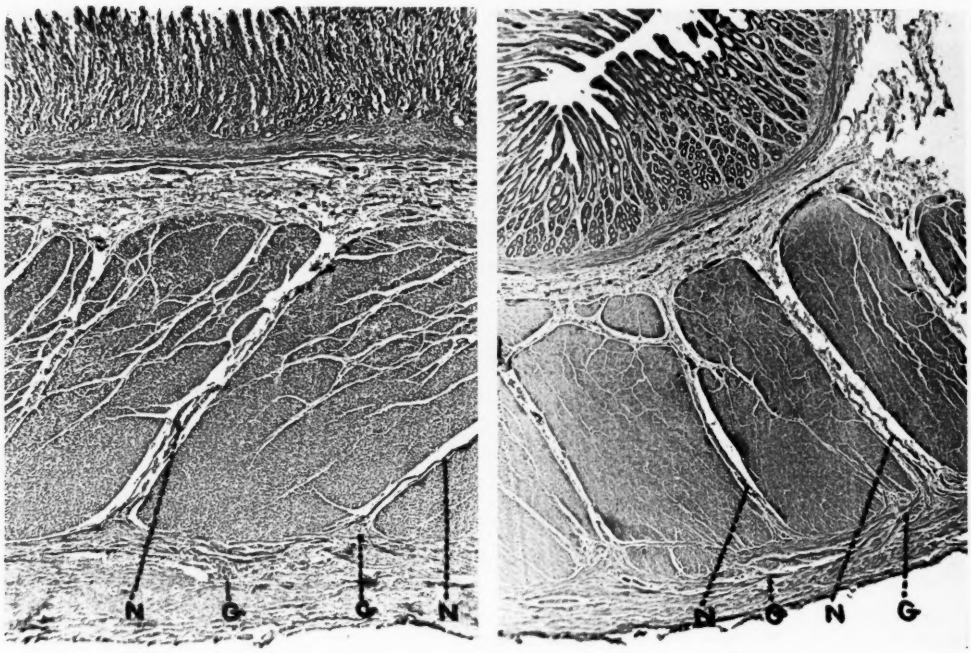
Fig. 2. Relationships of myenteric ganglion cells, nerve bundles and muscle thickness. a, In the normal pylorus of an infant, demonstrating an increase in all three over those seen in Figure 1a (cresyl violet, ×30). b, In pylorus of infant with hypertrophic pyloric stenosis, demonstrating the relative increase in thickness of circular muscle and relative decrease from normal in the number of ganglion cells and nerve bundles (cresyl violet, ×30).

actual amount of diminution of ganglion cells and nerve fiber bundles in the pylorus in hypertrophic pyloric stenosis. However, some diminution is readily apparent.

COMPARISON OF MORPHOLOGIC CHARACTERISTICS OF GANGLION CELLS OF THE MYENTERIC PLEXUS IN NORMAL STOMACHS AND DUODENUMS AND IN THOSE IN WHICH HYPERTROPHIC PYLORIC STENOSIS WAS PRESENT

No attempt was made in this study to distinguish between the types of ganglion cells as described by Dogiel and Hill because of the inconstancies of silver staining, and consequent inability to follow axons and dendrites.

In the normal infant and adult the myenteric ganglion cells in the stomach, pylorus, and duodenum are similar. The nucleus usually occupies from a fourth to a third of the cell, and the nuclear membrane distinctly outlines it from the surrounding cytoplasm. Generally one nucleolus is seen and the chromatin structure is fine with distinct fibrils radiating out from the nucleolus. The cytoplasm is normally medium to dark staining and is filled with Nissl's bodies, fibrils, and vacuoles (Figs. 4b and 5a to g).



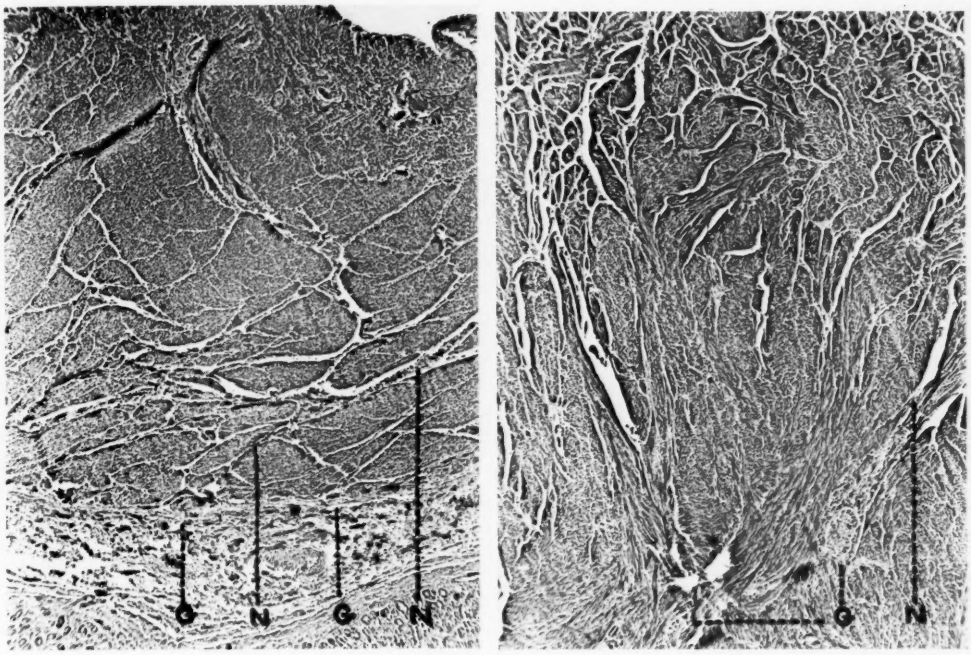
a b

Fig. 3. Relationships of myenteric ganglion cells, nerve bundles, and muscle thickness. a, In the normal adult stomach just below the incisura (cresyl violet, ×30). b, Similar location to that in, a, in an adult with persisting hypertrophic pyloric stenosis. This demonstrates little change from normal (cresyl violet, ×30).

As will be seen in Figure 5a to h, the ganglia in the stomachs above the pylorus of infants and adults with hypertrophic pyloric stenosis appeared normal, and only rarely was an indistinct cell seen. In the pylorus in hypertrophic pyloric stenosis, however, only an occasional ganglion cell appeared normal and the great majority had undergone change. The nuclear membrane was indistinct and the nucleolus appeared to be separated into two or three segments or to have disintegrated entirely. The cytoplasm in these cells for the most part was clear and light staining without fibrillar networks or vacuoles. Nissl's bodies were absent or indistinct. Often the cytoplasm appeared to be disintegrating with a loss of the cell membrane (Fig. 5d, e, and h). Numerous ghost cells were found in all sections of the pylorus in which the entire cell seemed to have lost its staining properties and merely appeared as a faint outline of a normal ganglion cell (Fig. 5e). These pathologic changes are similar to those seen by Ingersoll with excessive vagal stimulation and probably are due to exhaustion rather than any congenital defect in the ganglion cells such as is seen in congenital megacolon, described by Whitehouse, and Whitehouse and Kernohan.

COMPARISON OF MORPHOLOGIC CHARACTERISTICS OF MUSCULATURE IN NORMAL STOMACHS AND DUODENUMS AND IN THOSE AFFECTED BY HYPERTROPHIC PYLORIC STENOSIS

In cases of hypertrophic pyloric stenosis the circular musculature of the pylorus was two to four times as thick as that of the normal infants and often twice as thick as that of normal adults. However, the thickness of the circular muscle of the duodenum and stomach above the pylorus and the longitudinal muscle of all three areas remained



a b

Fig. 4. Relationships of myenteric ganglion cells, nerve bundles, and muscle thickness. a, In the normal pylorus of an adult demonstrating an increase in all three elements over those seen in Figure 3. b, In pylorus of adult with persisting hypertrophic pyloric stenosis demonstrating the relative increase in thickness of circular muscle and relative decrease from normal in the numbers of ganglion cells and nerve bundles (cresyl violet, ×30).

unchanged from the normal in the majority of cases of hypertrophic pyloric stenosis (Fig. 6). Microscopically the circular muscle of the pylorus in cases of hypertrophic pyloric stenosis has an irregular pattern with muscle fibers running in all directions. This irregular pattern resembles the pattern of a leiomyoma (Fig. 7). This disorganization of muscle fibers is not seen in the stomach above the stenosed area or in the duodenum of patients with hypertrophic pyloric stenosis and is not seen in the normal stomach, pylorus, or duodenum. It may represent hypertrophy of the longitudinal muscle fibers as they pass through the circular muscle although this could not be demonstrated. Some hypertrophy as well as hyperplasia was noted in all sections of individual circular muscle fibers of the pylorus in the presence of hypertrophic pyloric stenosis. This change was described previously by Hutchison. It is associated with flattening and elongation of the nuclei probably due to compression.

We found, as Horton did, a definite separation or block of fibrous tissue between the circular muscle of the pylorus and that of duodenum, but the longitudinal muscle is continuous (Fig. 6) in both normal stomachs and in those in which hypertrophic pyloric stenosis is present.

Like Horton we also found that segments of longitudinal muscle were absent from the pyloruses of infants. This was observed in specimens from normal infants as well as from those with hypertrophic pyloric stenosis.

COMMENT

As has been pointed out earlier in this paper, the theories concerning the causative factors in hypertrophic pyloric stenosis have

TABLE II.—TYPE OF MATERIAL AND GRADING OF FINDINGS

Material from stomachs	Cases	Myenteric ganglion cells*			Nerve fiber bundles*			Relative muscle thickness†		
		Stomach	Pylorus	Duodenum	Stomach	Pylorus	Duodenum	Stomach	Pylorus	Duodenum
Infants Normal stomachs	1	2	4	2	2	4	2	1	2	1
	2	2	3	1	3	3	1	2	3	1
	3	2	4	2	2	3	2	2	4	1
	4	2	3	1	2	3	1	3	3	1
	5	2	3	2	2	3	2	2	3	2
	6	1	3	1	1	2	1	2	3	1
	7	2	3	2	3	3	2	2	3	1
	8	2	4	1	3	4	1	2	4	1
	9	2	3	1	2	3	1	2	4	1
	10	2	3	1	1	3	2	2	4	1
	Average	1.9	3.3	1.4	2.1	3.1	1.5	2.0	3.3	1.1
Hypertrophic pyloric stenosis	11	2	3A‡	2	2	4	3	2	4	1
	12	1	2A	3	1	2	3	2	4§	2
	13	2	1A	3	2	2	3	2	4§	1
	14	2	2A	3	2	2	3	3	4	2
	15	3	2A	3	3	2	3	2	4§	2
	16	3	3A	2	2	2	2	2	4	1
	17	2	3A	3	2	2	3	2	4§	1
	18	3	1A	2	2	2	2	2	4§	1
	19	2	2A	2	2	1	2	2	4§	1
	Average	2.2	2.1A	2.5	2.0	2.1	2.6	2.1	4§	1.3
Adults Normal stomachs	20	2	4	2	3	4	1	3	4	2
	21	2	3	2	2	3	2	2	4	1
	22	2	4	2	2	3	2	3	4	2
	23	2	3	2	2	3	2	2	4	2
	24	2	3	2	2	3	2	2	3	2
	Average	2.0	3.4	2.0	2.2	3.2	1.8	2.4	3.8	1.8
Hypertrophic pyloric stenosis	25	3	2A	2	3	2	2	3	4§	2
	26	2	2A	2	2	2	2	3	4§	2
	27	2	3A	3	2	2	3	3	4§	2
	28	2	1	2	2	2	2	3	4§	2
	29	3	3A	3	3	2	3	2	4§	2
	Average	2.4	2.2	2.4	2.4	2.0	2.4	2.8	4§	2.0

*Grading here signifies: 1, occasional; 2, few; 3, frequent; 4, numerous.
†Grading here signifies: 1, normal thickness of infant duodenal muscle; 2, twice the thickness of 1; 3, three times the thickness of 1; 4, four times the thickness of 1.
‡A—Numerous atrophic ganglia seen.
§More than four times as thick as normal duodenum of infants. In some cases as much as eight times as thick.

been many and varied. The neurogenic imbalance theory has many adherents but little specific data have been accumulated to substantiate any tenets in this line of reasoning. It certainly seems logical to assume that the separate and independent vagal innervation (which appears to be the only motor innervation) of the pylorus, demonstrated by McCrea and Kuntz, the independent initiation of gastric emptying peristaltic waves (which are purely vagal in origin as shown by Hightower and Code—8, 25), and the narrowing of the

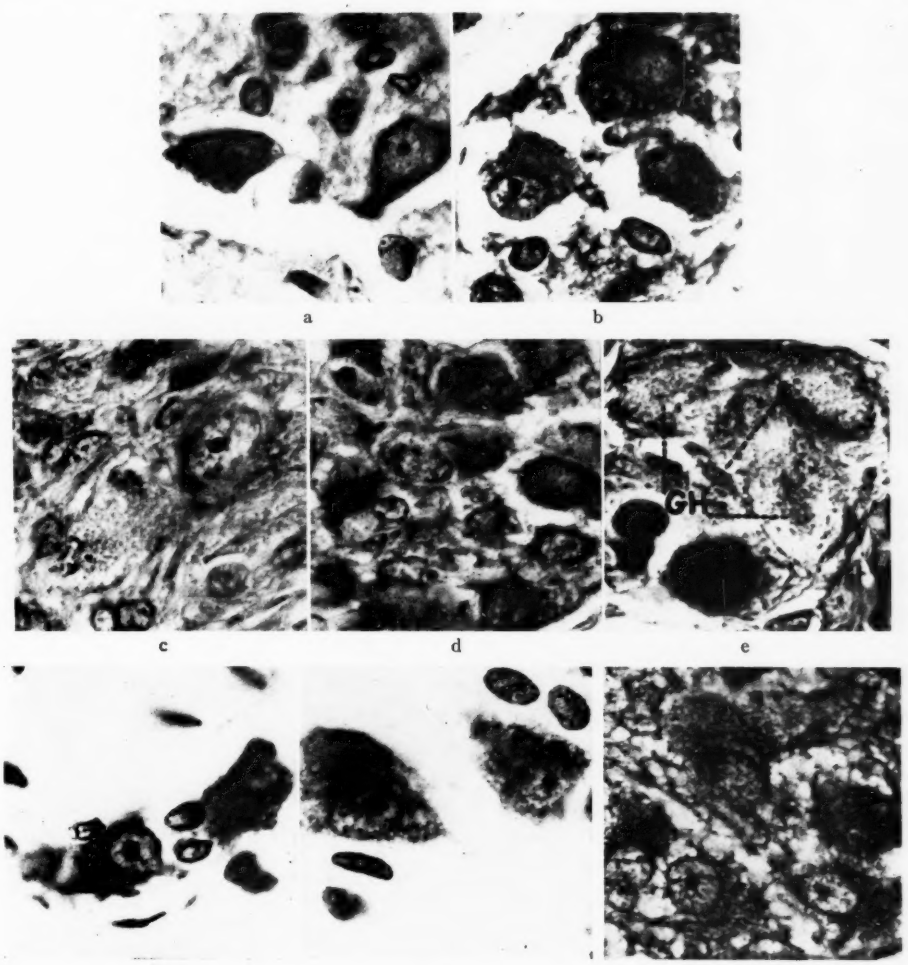


Fig. 5. Myenteric ganglion cells. a and b, In the infant (cresyl violet, ×880): a, normal, from the stomach just below the incisura in hypertrophic pyloric stenosis; b, from the normal pylorus. c, d, and e, From the pylorus of the infant with hypertrophic pyloric stenosis (cresyl violet; a and d, ×880; e, ×800): c, numerous degenerating cells, loss or fragmentation of nucleoli and nuclear membrane and loss of clear cytoplasm outline and fibrillar network; d, dark-staining cytoplasm, loss of cytoplasmic outline and fibrillar network and loss of normal nucleolar chromatin pattern; e, ghost cells (*GH*). f and g, From the adult (cresyl violet, ×800): f, normal from stomach just below the incisura in persisting hypertrophic pyloric stenosis; g, from normal pylorus. h, From the pylorus of the adult in persisting hypertrophic pyloric stenosis showing similar degenerative changes to those seen in the infant. Numerous ghost cells are seen here (cresyl violet, ×880).

pyloric outlet of the stomach would readily predispose to hypertrophy of the circular muscle of the pylorus in cases of excessive vagal stimulation. This excessive stimulation might or might not be the result of nervous inco-ordination between the segments of the stomach.

The changes in the ganglion cells of the myenteric plexus in the pylorus, as described previously in this paper, closely resemble

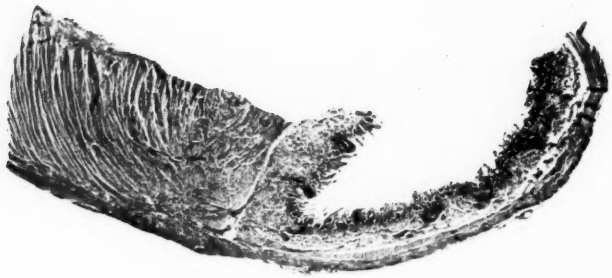


Fig. 6. The thickness of the muscle in the stomach, pylorus, and duodenum, the fibrous block between circular muscles of the pylorus and duodenum and the continuity of the longitudinal muscle of the duodenum and pylorus (hematoxylin and eosin, ×6).

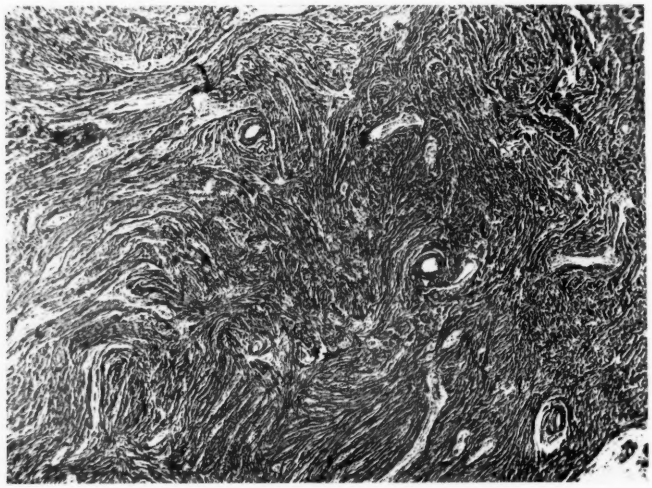


Fig. 7. The leiomyomatous structure of the pyloric circular muscle in hypertrophic pyloric stenosis (hematoxylin and eosin, ×35).

those changes described by Ingersoll after excessive vagal stimulation. It is unlikely that such changes may occur as a result of atrophy or disuse secondary to primary hypertrophy of the circular muscle. This primary hypertrophy was considered by Hirschsprung to be a primary congenital abnormality. In our opinion this is unlikely because Hill has shown that all impulses to individual muscle fibers originate in the myenteric ganglia, and so the ganglion cells would have had to be active at some time to cause the hypertrophy.

The evidence, as shown by Bendix and Necheles, that patients with hypertrophic pyloric stenosis as infants have a high incidence of duodenal ulcer in later life suggests also a vagotonic state in these particular individuals.

The theory that work hypertrophy due to primary muscular anomalies of the pylorus is a cause of hypertrophic pyloric stenosis is based on the observation that some segments of longitudinal muscle (the muscle which opens the pylorus) are missing. However, as stated previously in this study, the pylorus of the normal infant shows the same absence of segments of longitudinal muscle. This indicates that the pyloric circular muscle in hypertrophic pyloric stenosis is probably not hypertrophic because it is working against a pylorus which fails to open. The reason that longitudinal muscle fails to hypertrophy to the same extent as the circular muscle in hypertrophic pyloric stenosis is probably because nerve impulses are distributed as readily and as equally to the duodenum as to the pylorus, since no block in this musculature occurs at the pyloroduodenal junction and because fewer nerve fiber tracts run from the myenteric plexus of the pylorus to the longitudinal muscle in this area.

It is interesting to speculate why a longitudinal incision through the circular muscle layer of the pylorus in hypertrophic pyloric stenosis, as is performed in the Ramstedt operation, will cause the hypertrophy of the pyloric circular muscle to disappear and the muscle to regain its normal gross and microscopic appearance within a matter of months, as demonstrated by Donovan and Holt. This is rather difficult to explain if the neurogenic theory is accepted as the explanation of the cause of hypertrophic pyloric stenosis unless the incision interrupts enough of the vagal fibers and fibers of the myenteric plexus so that diminution of the vagal stimuli will allow restoration of the ganglia and muscle to normal.

SUMMARY AND CONCLUSIONS

The ganglion cells and nerve fibers of the myenteric plexus and the musculature of normal stomachs and duodenums and also of those affected by hypertrophic pyloric stenosis were studied. Findings in normal stomachs of 10 infants and 5 adults were as follows:

1. The number of ganglion cells and nerve fiber tracts of the myenteric plexus per unit area of muscle tissue increases progressively from a point just below the incisura of the stomach down through the pylorus to its junction with the duodenum.

2. The duodenum contains fewer myenteric ganglion cells and nerve fiber bundles per unit area than the stomach just below the incisura. This decrease is sharply apparent at the junction of the duodenum and pylorus.

3. The thickness of the musculature is increased from a point just below the gastric incisura down to the pyloroduodenal junction. This increase consists mostly of increase in thickness of the circular muscle. Abruptly at this point the duodenal musculature becomes only about half as thick as the gastric muscle just below the incisura.

4. The myenteric ganglion cells in the stomach, pylorus, and duodenum are almost identical morphologically and practically no degenerating ganglion cells are seen in any of these segments.

In the stomach, pylorus, and duodenum of infants and adults who had hypertrophic pyloric stenosis the following observations were made:

1. The number of myenteric ganglion cells and myenteric nerve fiber tracts per unit area appears to be reduced in the pyloric region but remains quantitatively normal or slightly increased in the stomach just below the incisura and in the duodenum.

2. The circular muscle of the pylorus of both infants and adults is two to four times as thick as that of normal infants while the thickness of the circular muscle of the remainder of the stomach, pylorus, and duodenum remains normal or only slightly increased. Individual fibers of the circular muscle in the pylorus show evidence of hypertrophy and perhaps hyperplasia with flattening of the nuclei.

3. The most significant and constant finding in our study was that the majority of myenteric ganglion cells in the pylorus showed degenerative changes resembling those seen after excessive vagus nerve stimulation. Such changes are

not seen in the myenteric ganglion cells in the stomach above the pylorus nor in the duodenum in pyloric stenosis or in normal individuals. These changes should be sharply contrasted with the congenital absence of myenteric ganglion cells found in the so-called stenosed area in congenital megacolon.

The following observations were made in both the normal stomachs, duodenums, and pyloruses of infants and adults and also in those of infants and adults affected by hypertrophic pyloric stenosis:

1. Segments of longitudinal muscle are often found to be absent in the pylorus of normal infants as well as in the pylorus of those infants who have hypertrophic pyloric stenosis.

2. There is a definite fibrous block between the circular musculature of the pylorus and that of the duodenum while the longitudinal muscle is continuous in the two segments.

3. The circular muscle hypertrophy in infants with hypertrophic pyloric stenosis persists into adulthood if gastroenterostomy alone is performed in infancy.

Our findings lend support to the neurogenic theory of the etiology of hypertrophic pyloric stenosis.

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THE MYENTERIC PLEXUS IN CHRONIC ULCERATIVE COLITIS

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IN 1938, Robertson and Kernohan reported the absence of ganglion cells in the myenteric plexus of the lower portion of the colon in a case of congenital megacolon. For comparison they studied the myenteric plexuses in 2 cases of chronic ulcerative colitis, because the latter was a clinical entity that was the very antithesis of congenital megacolon, namely overactivity. They found a great increase in ganglion cells in the myenteric plexus in chronic ulcerative colitis. However, a question remained as to whether this increase was actual or only relative, and whether it was caused by the pathologic contracture of the colon that is seen especially in advanced chronic ulcerative colitis.

The purpose of this study was to make histologic examinations of the myenteric plexuses in colons affected by chronic ulcerative colitis and to compare the findings with those in normal colons, particularly in reference to the quantity of ganglion cells present.

ANATOMY AND PHYSIOLOGY

Search of the literature failed to elucidate this problem. Wail described the histopathologic changes of nerves in the myenteric plexus in experimental chronic ulcerative colitis but made no mention of increased numbers of ganglion cells.

Excellent reviews concerning the innervation of the digestive tract have been written by Hill, Schabadasch, and Alvarez. Much of this work has been done on animals. The nerve supply to the colon consists of extrinsic and intrinsic systems.

Extrinsic nerves. The extrinsic nerves are composed of motor parasympathetic and inhibitory sympathetic fibers that enter the colon from without.

Parasympathetic innervation of colon.—Most investigators considered that the vagal fibers innervate the colon for a variable distance from the ileocecal valve but that they usually terminate within the first half or third of the transverse colon. The pelvic nerves (nervi erigentes) from the sacral parasympathetic outflow, supply the remainder of the colon and rectum. A few investigators were of the opinion that the vagus nerve does not supply any of the large intestine.

Sympathetic innervation of colon.—Sympathetic fibers arise from the lower thoracic and the lumbar segments of the spinal cord as preganglionic fibers. They synapse in either the vertebral ganglia or in the mesenteric ganglia and reach the colon as postganglionic fibers. The nerves to the proximal part of the colon travel along the superior mesenteric vessels; those to the distal portion of the colon travel largely by way of the inferior mesenteric and superior hemorrhoidal vessels.

Relationship of parasympathetic and sympathetic fibers to enteric ganglia.—The vagus and pelvic nerves are composed of preganglionic parasympathetic fibers that synapse with the ganglion cells of the enteric plexus. The sympathetic fibers reaching the colon are all postganglionic. They do not synapse in the enteric plexus but terminate in the structures innervated. Thus it would appear that the ganglion cells of the myenteric plexus belong to the parasympathetic system.

Some morphologists considered that the neurons in the enteric plexus may be either sympathetic or parasympathetic in origin. Van Campenhout, working with chick embryos, was of the opinion that the enteric plexus is sympathetic in origin.

Intrinsic nerves. The intrinsic system is composed of two well defined plexuses: the

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TABLE I.—LOCATION IN COLON AND DIRECTION IN WHICH BLOCKS OF TISSUE WERE TAKEN

Location in colon	Direction of section	Blocks, No.	
		Chronic ulcerative colitis*	Controls
Rectosigmoid	Transverse Longitudinal	25 7	25 4
Upper part of sigmoid	Longitudinal	7	1
Descending	Transverse Longitudinal	25 7	25 4
Transverse	Longitudinal	7	1
Ascending	Transverse Longitudinal	25 7	25 4

*In 2 additional cases 16 blocks were made. These were not included in the results because of excessive destruction of myenteric plexus. In 1 additional case 6 blocks were made. This case also was excluded because of the short duration of disease (subacute ulcerative colitis).

myenteric (Auerbach's) and the submucosal (Meissner's). Stöhr, Lawrentjew and Penfield described the intrinsic nerve supply in more detail. They listed four systems but mentioned that they are intimately connected. These systems are the subserous, myenteric, deep muscular, and submucosal. Schabadasch added a fifth system between the deep muscular and submucosal, namely the internal plexus of Henle.

Myenteric (Auerbach's) plexus.—Since the myenteric plexus is the most prominent of these systems and since the other plexuses are probably dependent on it, this study has been confined to an examination of Auerbach's plexus.

The myenteric plexus is a network of interlacing bundles located between the outer longitudinal and inner circular layers of muscle. It consists of nerve cells collected in groups or ganglia, which are connected by nerve bundles that contain both extrinsic and intrinsic fibers. Stöhr and Schabadasch both described primary, secondary, and tertiary subdivisions of the myenteric plexus. The ganglion cells are almost entirely restricted to the primary division; they exhibit the usual variation in size and form according to the position they occupy in the intestinal wall and the plane in which they are cut.

In addition to nerve elements of the myenteric plexus, numerous supporting cells are present that form a dense feltwork around the ganglion cells and nerve fibers. These supporting cells are smaller than ganglion cells; their shape varies from oblong or triangular, to elongated and fusiform. The supporting cells have a relatively small amount of visible cytoplasm that frequently has a vacuolated appearance. The nucleus is usually oval in form; it has no prominent nucleolus and the granules of chromatin are scattered throughout the reticulum. From the body of the cell arise two or more unevenly dilated processes. Some anatomists consider these cells to be nervous tissue but most believe they are connective tissue.

We have seen other small cells in the myenteric plexuses of most persons that may be underdeveloped nerve cells. Stöhr claimed that, in addition to Dogiel's nerve cells of types 1 and 2 (2), many small and middle-sized cells are found that are not greatly different from ganglion cells. However, thus far it has not been established definitely that these cells are true nerve cells.

The main functions of the myenteric plexus are to expedite conduction and to co-ordinate the activities of muscular fibers. An important function of the ganglion cell is probably to keep the muscle from contracting into a hard knot and staying that way (cardiospasm, Hirschsprung's disease). The nerves of the intestine also serve to keep the muscle from responding violently to every stimulus that comes along.

In general, stimulation of the parasympathetic nerves to the colon excites peristalsis and secretion, whereas stimulation of the sympathetic nerves inhibits peristalsis and secretion. Exceptions are noted, apparently owing to physiologic states and to the assumed presence of some inhibitory fibers in the parasympathetic nerves, and of some excitatory fibers in the sympathetic nerves.

MATERIALS AND METHODS

Twenty-five cases of chronic ulcerative colitis and 25 controls were selected for study. The controls were cases in which necropsies were done and in which the colons were clinically, grossly, and microscopically normal. The controls were taken in order except that an attempt was made to have patients in a similar age group to the patients who had

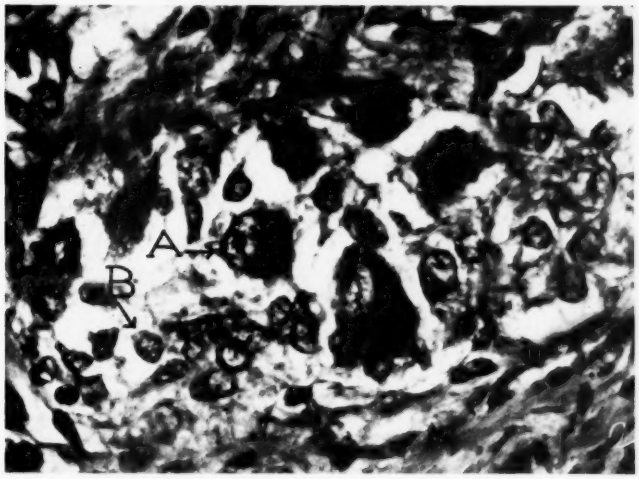


Fig. 1. Transverse section. Ganglion cells from descending colon of chronic ulcerative colitis specimen (hematoxylin and eosin; ×600). *A*, Ganglion cell; *B*, supporting cell. The cresyl violet staining of supporting cell in Figure 2a should be compared.

hronic ulcerative colitis. Both diffuse and egmental types of chronic ulcerative colitis ere represented in this study. The diseased olons were obtained both at necropsy and at peration. In the diffuse type, involvement of he entire colon was present, beginning at the nus, while in the segmental type, only parts of the colon were diseased. The duration of disease was 4 years or more in most instances. An attempt was made to include some patients who were less than 20 years of age and some who were more than 40. No cases in which carcinoma was present were included. One case of subacute diffuse ulcerative colitis was encountered; the findings in this case were of interest but are not included in the over-all statistics.

Two cases were rejected because of excessive destruction and distortion of the myenteric plexus by inflammation. In nearly all other cases, the myenteric plexus was remarkably well preserved in spite of severe inflammation of the intestinal wall elsewhere.

Table I indicates the locations from which blocks of tissue were taken. All blocks were from specimens fixed in formalin and were embedded in paraffin; they totaled 221 in all. Sections were all 8 microns thick and were cut in longitudinal or transverse directions, perpendicular to the intestinal surface.

The first major problem was to determine which staining methods would be best for outlining ganglion cells. We were not especially concerned with nerve fibers.

The best histologic studies of the myenteric plexus have been made using supravital staining methods in tissues removed from animals. However, this method is not applicable to the study of formalinized specimens. The following stains were used: hematoxylin and eosin, van Gieson, modified Bodian, and cresyl violet. Hematoxylin and eosin and van Gieson's stain were found to be of general value but were not specific for nerve cells (Fig. 1). The modified Bodian method of impregnation with silver showed nerve fibers and ganglion cells rather well. Cresyl violet was excellent for the study of nerve cells. The ganglion cells stained much more readily than the adjacent supporting cells and thus stood out in bold relief (Fig. 2). Therefore, we decided to stain all sections with hematoxylin and eosin and with cresyl violet; a smaller number was stained by means of the modified Bodian technique.

The second major problem concerned the best way to measure the myenteric plexus.

TABLE II.—COMPARISON OF CLINICAL AND LABORATORY FINDINGS IN 25 CASES OF CHRONIC ULCERATIVE COLITIS AND 25 CONTROLS

		Colitis	Controls
Age, years	Average	33.9	53.2
	Range	15 to 69	14 to 85
Sex	Male	12	17
	Female	13	8
Source of material	Necropsy	11	25
	Operation	14	0
Duration of disease, years	Average	8.3	
	Range	1.4 to 17.0	
Type of colitis	Diffuse	17	
	Segmental	8	
Length of colon, cm. (formalinized)	Average	76	150 (not formalinized)
	Range	60 to 90	

TABLE III.—AVERAGE COLONIC CIRCUMFERENCE IN CENTIMETERS IN 25 CASES OF CHRONIC ULCERATIVE COLITIS AND 25 CONTROLS

Location	Colitis	Controls
Rectosigmoid	4.45± 0.29*	5.04± 0.30
Descending colon	4.57± 0.31	5.42± 0.30
Ascending colon	5.08± 0.25	6.72± 0.54

*Values after the ± are standard errors of the means.

TABLE IV.—AVERAGE COUNTS OF GANGLION CELLS IN TRANSVERSE SECTIONS OF THE COLON IN 25 CASES OF CHRONIC ULCERATIVE COLITIS AND 25 CONTROLS

Location of block	Colitis		Controls	
	Cells per cm.	Cells in circumference, estimated total	Cells per cm.	Cells in circumference, estimated total
Rectosigmoid	52.2± 4.1*	215.4	17.6± 1.9	82.6
Descending colon	47.4± 5.2	203.5	13.9± 1.2	73.0
Ascending colon	39.2± 2.8	194.8	10.5± 1.0	63.9

*Values after the ± are standard errors of the means.

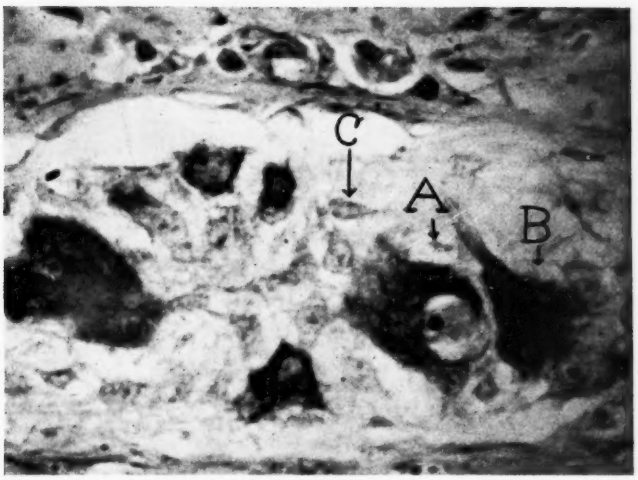
TABLE V.—COMPARISON OF COLONS IN DIFFUSE AND SEGMENTAL TYPES OF CHRONIC ULCERATIVE COLITIS

Location of block	Average of 17 cases of diffuse chronic ulcerative colitis			Average of 8 cases of segmental chronic ulcerative colitis		
	Circumference, cm.	Ganglion cell count		Circumference, cm.	Ganglion cell count	
		Per cm.	In circumference, estimated total		Per cm.	In circumference, estimated total
Rectosigmoid	3.96	58.5	220.5	5.49	38.7	204.5
Descending colon	4.08	52.0	202.6	5.61	37.5	205.4
Ascending colon	4.78	42.9	197.6	5.70	32.7	188.9

Two chief methods became apparent at once, namely to count the myenteric-plexus bundles and to count ganglion cells. Counting the bundles was discarded because their size was so variable, because such counts did not represent the amount of functioning nerve elements (some bundles had no ganglion cells), and because in some sections the bundles were cut along their longitudinal axes for long distances. Therefore, it was decided to count individual ganglion cells in transverse sections only for quantitative results.

At first the ganglion cells of the entire circumference were counted, but it soon became apparent that a strip measuring 2 centimeters in length was representative. At least 1 taenia coli was included in each section because Irwin found an increase in ganglion cells under the taeniae in guinea pigs and because Lium and Porter found the most severe ulcerations to be under the taeniae in ulcerative colitis. Ganglion cells were counted for 2 centimeters of the circumference; half this number represented the cells per centimeter. The estimated total number of cells for a specific transverse level was obtained by multiplying the number of cells per centimeter by the circumference of the colon in centimeters. In a few cases it was necessary to count the nerve cells in 1 centimeter instead of 2 centimeters.

It was decided to count only the ganglion cells that contained both nucleus and nucleolus, as this made for greater accuracy in identification and count (Fig. 2). In several slides all ganglion cells were counted whether they contained a nucleolus or not in order to observe the total number of nerve cells in a section. A ganglion cell was not counted if doubt existed as to its identity. No attempt was made to differentiate cells as to whether



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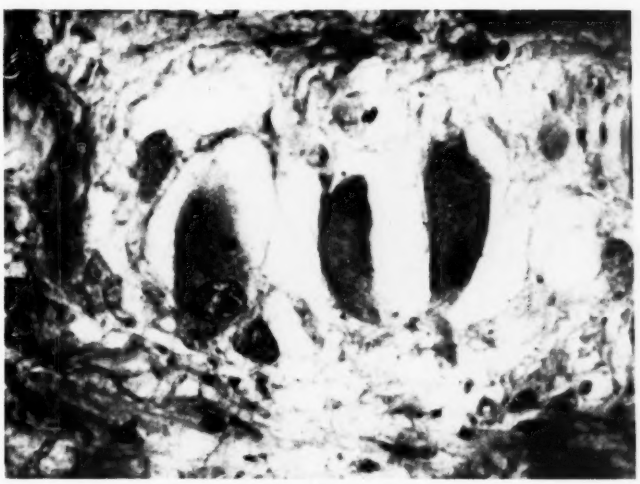

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Fig. 2. a, Ganglion cells from descending colon in chronic ulcerative colitis. *A*, Ganglion cell with nucleolus; *B*, ganglion cell that does not demonstrate nucleus and nucleolus; *C*, supporting cell. Note great affinity of stain for ganglion cells (cresyl violet; ×600); b, ganglion cells from descending colon in chronic ulcerative colitis (modified Bodian, ×600).

they were Dogiel's type 1 or 2, nor were cells scrutinized closely for degenerative changes.

The following clinical and pathologic data were tabulated in each case of ulcerative colitis: age, sex, type of chronic ulcerative colitis, duration of disease, results of proctosigmoidoscopic examination and colonic roentgenogram, pathologic report, length and circumference of colon, nerve-cell counts at various levels, and whether or not an increase of nerve cells under the taeniae was present.

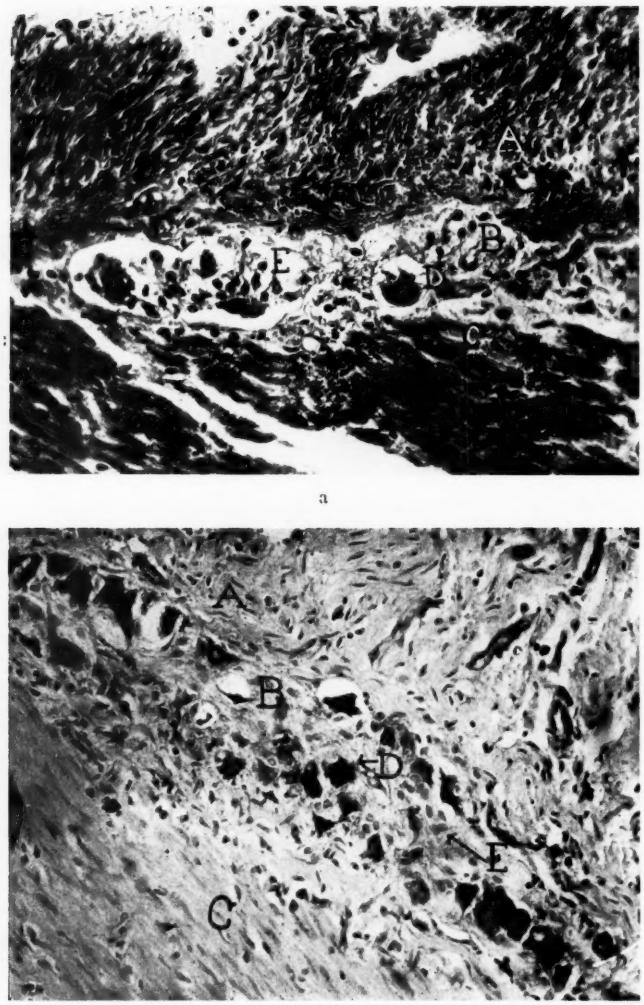


Fig. 3. a, Ganglion cells from rectosigmoidal level of normal colon; b, ganglion cell from descending colon from patient who had chronic ulcerative colitis; note great increase in ganglion cells as compared to a. *A*, Longitudinal muscle; *B*, myenteric plexus; *C*, circular muscle; *D*, ganglion cell; *E*, supporting cell (both cresyl violet, ×200).

RESULTS

Tables II and III show some of the clinical and pathologic data. From these tables it is evident that the colons affected by colitis underwent shrinkage both circumferentially and longitudinally. Circumferential shrinkage is less pronounced in the segmental type of chronic ulcerative colitis than in the diffuse type. The colon that is placed in formalin undergoes a shrinkage of about 15 to 20 per cent. In consideration of this factor, the colons affected by colitis in this study measured

slightly less than two-thirds the length of a normal average colon. This is in agreement with Warren and Sommers, who noted a shrinkage of one-third in fresh specimens of colons that were affected by chronic ulcerative colitis.

It is apparent from Table IV that a great increase took place in the number of ganglion cells in the colons of patients who had chronic ulcerative colitis; the actual number of cells per centimeter was a little more than three times that seen in normal colons, and the estimated total count was a little less than three times the normal count (Fig. 3). Even if it is considered that the pathologic contracture of one-third can concentrate the cells there is still a twofold increase in ganglion cells. These findings are depicted in Figure 4.

Sections taken from the rectosigmoid, the descending colon, and the ascending colon exhibited, in the order named, a steady decrease in the number of ganglion cells in cases of colitis and also in controls.

The number of cases in which ganglion cells were increased under the taeniae was in the minority, namely 17 per cent and 21 per cent in colons affected by chronic ulcerative colitis and in normal colons respectively.

An attempt was made to determine what relationship, if any, existed between the number of cells in the myenteric plexus and such factors as the type of chronic ulcerative colitis, the duration of disease and the age and sex of the patient. Although some of the subgroups contain too few cases to allow definite conclusions, they do indicate the general trend.

From Table V it can be seen that the essential difference between colons affected by diffuse and by segmental types of chronic ulcerative colitis is the consistently greater circumference of the segmentally diseased colons at all levels examined. Circumferences of the latter are similar to those of normal colons, which probably accounts for the lack of much difference in circumference between colons affected by chronic ulcerative colitis and normal colons at the rectosigmoidal level. In most cases, the rectosigmoidal areas were uninvolved in the segmental type of chronic ulcerative colitis. The number of ganglion cells

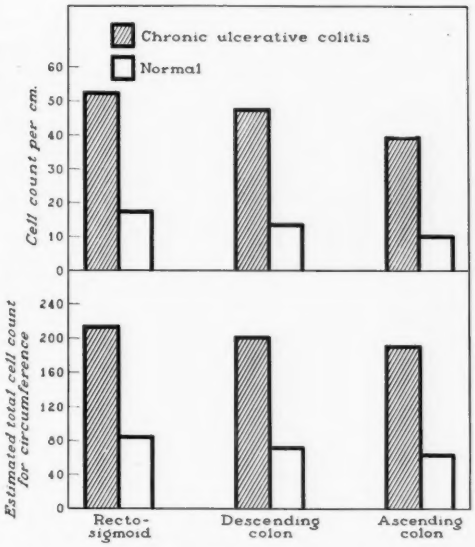
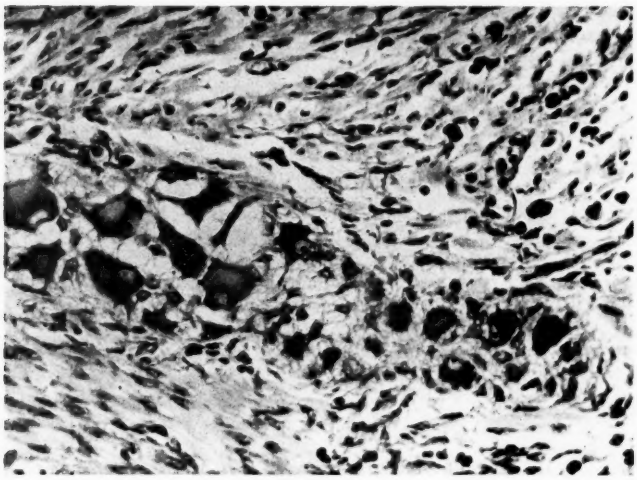


Fig. 4. Actual and estimated ganglion cell counts from various regions of the colon.

per centimeter was naturally less in the latter form of disease but the estimated total number of nerve cells was essentially the same in these two types of chronic ulcerative colitis. The number of ganglion cells in uninvolved portions of the colon in the segmental type of disease was usually slightly greater than the number in the adjacent involved segments; the number of cells was also always greater in the uninvolved segments of segmentally diseased colons than in correspondingly located segments of normal colons.

The cases of chronic ulcerative colitis were divided in two groups, namely those in which the disease was less than 4 years in duration and those in which it was more than 4 years in duration. No significant difference was present in the number of cells in these two groups. The disease in the previously mentioned case of subacute ulcerative colitis had been present for 3 months. The circumferential measurements of the colon were greater at all levels than those of the average normal colon; the longitudinal shrinkage was only about one-sixth. The number of ganglion cells per centimeter was slightly less than in cases of chronic ulcerative colitis but was con-



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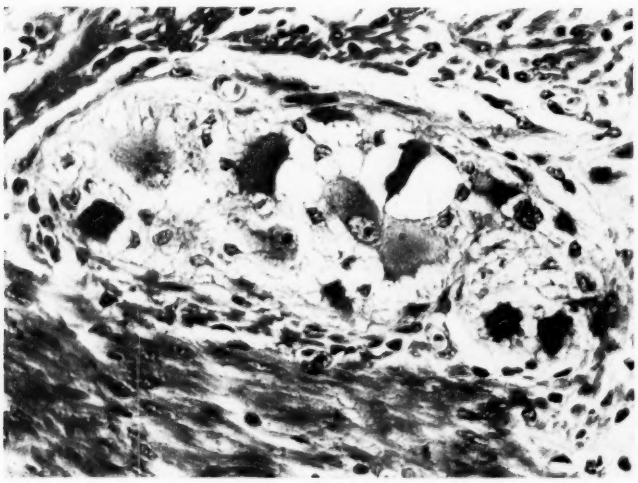

b

Fig. 5. Ganglion cells from descending colon in subacute ulcerative colitis. a, Note great increase in number of ganglion cells during the stage of minimal pathologic contracture; note also the reaction of inflammation (cresyl violet, ×280). b, Note chromatolysis and great increase in number of ganglion cells (hematoxylin and eosin, ×340).

siderably greater than that noted in the normal colon; the estimated total ganglion cells in the circumference was even greater than in cases of chronic ulcerative colitis (Fig. 5).

Patients who had colitis were separated on the basis of age; a comparison was made between those who were less than 20 years of age and those who were more than 40. The only difference was at the rectosigmoidal level. The circumference of this part of the colon was appreciably less in those more than 40 years of age; patients in this group had a

concomitant increase in the number of ganglion cells per centimeter. The estimated total number of ganglion cells, however, was similar in both groups.

No significant difference in the sexes was noted except that the circumference of the descending colon in females averaged somewhat less than in males. However, the number of ganglion cells per centimeter was increased in females, thus making no difference in the estimated total number of cells.

The ganglion cells ranged from 30 to 60 microns in diameter. The supporting cells were 15 to 20 microns in diameter. The ratio of all identifiable ganglion cells to cells with nucleoli varied from 2:1 to 2.5:1. In general, longitudinal sections of the colon did not outline the myenteric plexus as well as did transverse sections and the cell counts were slightly less at a given level of the colon in the former than they were in the latter.

The nerve cells of the myenteric plexus usually appeared to be spared the extensive inflammation of other tissues in the colon. However, at times some destruction of nerve cells was present so that, if anything, the number of nerve cells was underestimated.

SUMMARY

The number of ganglion cells in the myenteric plexuses at various levels in the colon in 25 cases of chronic ulcerative colitis was compared to those in a like number of normal colons. The colons affected with chronic ulcerative colitis were obtained at necropsy or at operation and represented both the diffuse and segmental types of disease.

Approximately a threefold increase was found in the number of ganglion cells in cases of chronic ulcerative colitis. Even when the pathologic contracture seen in ulcerative colitis is taken into consideration, an actual twofold increase in ganglion cells was noted in this disease.

The number of ganglion cells per centimeter in the segmental type of chronic ulcerative colitis was less than in the diffuse variety. However, the circumferential shrinkage of the segmental type was substantially less and, consequently, the estimated total number of ganglion cells was the same. The number of ganglion cells in the uninvolved portions in segmental types of chronic ulcerative colitis was usually slightly greater than in the adjacent involved segments.

Duration of disease and age and sex of the patient had no significant influence on the number of ganglion cells. In 1 case of subacute ulcerative colitis of only 3 months' duration, a great increase in ganglion cells already was present.

No obvious explanation exists for this increase in the number of ganglion cells in chronic ulcerative colitis. It may be that the stimulus of ulceration and increased function of the colon causes these so-called small, underdeveloped nerve cells to enlarge and to become adult in type and function. On the other hand, it is possible but improbable that certain persons are endowed with more than the average amount of myenteric-plexus tissue and that chronic ulcerative colitis may develop in some of these persons. Little or no evidence can be adduced to show that nerve tissue is able to increase after birth.

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AN INCISIONAL APPROACH FOR CHOLECYSTECTOMY AND CHOLEDOCHOTOMY DESIGNED TO REDUCE INJURIES TO THE COMMON DUCT

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A HIGHLY satisfactory experience with an old but recently rediscovered incision for exposure of the gallbladder and common duct, fully described by Kehr in 1913, warrants a detailed description of this excellent approach to an important surgical area. Apparently overlooked or ignored, Kehr's incision has not been described in American textbooks of operative surgery. That it is worthy of greater consideration can be demonstrated fully by a trial use. The directness of the approach and the resulting improved exposure of vital structures should reduce materially the accidental injuries to the common duct. In passing, it should be stated that the number of operative injuries to the common duct requiring complicated, difficult, and frequently disappointing operations for their correction has become truly appalling. They are a serious indictment, first, of the morality of our medical graduates who dare do that for which they have had no training, and, second, of the laxity of our licensure laws that permit a recently graduated doctor of medicine or of osteopathy to put up a sign "Physician *and Surgeon*," which enables him without adequate training to inflict a destructive scalpel upon an unsuspecting public. Unhappily, the untrained surgeon is occasionally matched by the bold and overconfident operator who through an assumed superior familiarity works with too great haste, or becomes casual in his precautions, or operates as Sir Frederick Treves once remarked upon "the canine principle of savage attack." Manifestly, the moment a surgeon becomes casual or reckless, the life or future comfort of his patient is endangered. In surgery of the biliary tract the careless or poorly trained operator is truly a menace.

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For simple cholecystectomy, the Kehr incision runs in the midline (Fig. 1a) from the xiphisternum halfway to the umbilicus, turns obliquely to the right to parallel the midcostal margin, transecting the right rectus muscle completely (Fig. 1b). Occasionally it is necessary to extend the incision somewhat more laterally through the oblique and transverse muscles, but usually injury to the tenth intercostal nerve is avoided, an important advantage over the usual straight subcostal incision. The round ligament of the liver is divided, and the vessel invariably accompanying it is controlled by ligation.

The midline component through the linea alba appears to release the costal margin permitting its easier retraction upward and outward to provide an exposure of the common duct area rarely achieved by the usual subcostal or right rectus incision. The second assistant has repeatedly remarked upon the increased facility with which he is able to observe what the operator is doing—a great boon to this long-suffering member of the operating team. Because of the high location of the Kehr incision, the small bowel rarely gets in one's way, and when the incision is closed, only stomach and large bowel lie beneath it, thus obviating to great extent the possibility of small bowel adherence and later obstruction, a distinct advantage over the long right rectus or paramedian incision.

The Kehr incision has proved surprisingly adequate for concomitant hemigastrectomy which was found necessary in 2 patients with serious duodenal ulceration accompanying gallstones. Slightly modified, the incision has been adapted also to operations for carcinoma of the pancreas: exploration of the abdomen is first performed through the short upper midline incision; if absence of metastases to the liver or peritoneum suggests that resec-

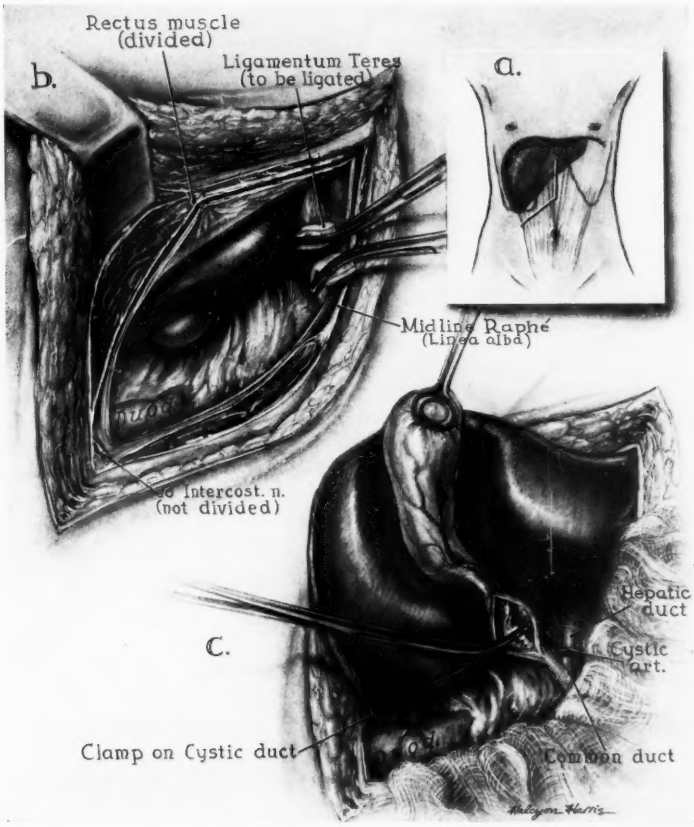

Fig. 1. a and b, The Kehr incision which lies partly in midline with oblique transection of right rectus muscle permits upward and lateral displacement of costal margin for a more direct approach to the common duct area. c, The first step in cholecystectomy is identification of common and cystic ducts for application of a clamp to the cystic duct to prevent displacement of stones into common duct during manipulation of the gallbladder.

tion of the pancreas may be feasible, the incision is extended obliquely to the right, paralleling the costal margin with transection of the right rectus muscle. If the lesion is now found inoperable except for correction of the jaundice, ample exposure is available for a palliative cholecystjejunostomy and jejunojejunostomy. If resection of the pancreas should prove possible, the incision is then converted into an inverted Y by a second oblique extension from the caudad end of the midline incision paralleling the left costal margin with partial transection of the left rectus muscle.

Before proceeding with cholecystectomy, a general survey of the abdomen is made by palpation, or by inspection wherever possible, of the duodenum, pylorus, stomach, pancreas, large bowel, both kidneys, and the pelvic organs. Normal as well as abnormal findings are dutifully recorded in the operative note for future reference. The importance of this general survey cannot be stressed too strongly: in 1 instance a totally unsuspected carcinoma of the stomach was discovered and resected. This patient is well 6 years after operation. In 2 cases hemigastrectomy for severe duo-

denal ulceration was necessary. On another occasion, unsuspected bilateral adenocarcinomas of the ovaries were removed concomitantly through a second lower midline incision. If a mobile cecal mesentery permits *easy* delivery of the appendix, the latter is removed through the Kehr incision. If an appendectomy is definitely indicated and the appendix is not easily deliverable, it is removed through a separate McBurney incision. Attempts to deliver the appendix by excessive traction on the large bowel and cecum should be avoided since serious postoperative distention is an inevitable sequel.

The first step in the cholecystectomy is to identify accurately the cystic duct at its junction with the common duct and to isolate it sufficiently to apply a right-angled clamp (Fig. 1c) in order to prevent the accidental displacement of small stones into the common duct during operative manipulation. The fundus of the gallbladder is then completely mobilized before transecting the cystic duct (Fig. 2a). This permits: (1) accurate verification of the identity of the previously exposed cystic duct, (2) completely accurate identification of the common duct, and (3) accurate identification of the cystic and hepatic arteries. The latter artery, whose anatomic variations are more numerous than those of any other similarly important vessel in the body, may be anterior or posterior to the common duct, anterior or posterior to the cystic duct, making it highly vulnerable to injury by any haphazard moves in this dangerous area, as indicated by Michels and Moosman and Coller. "More damage can be done in a second's time than can be corrected in this patient's lifetime" is a truism that needs restatement and reiteration at each cholecystectomy for the benefit of the younger members of the team, and of the operator as well! As pointed out many times by many surgeons, accurate identification of each structure is absolutely necessary before applying a clamp or dividing any structure in this area. To this end, every effort should be directed to avoid flooding the field with blood by identifying and clamping the cystic artery before division. When blood from divided vessels wells up into the operative area, it is removed by *suction* rather than by sponges. This permits more precise identification and localization of the bleeding point and, therefore, more accurate application of a hemostat. The hurried, precipitate, and blind groping for control of a bleeding vessel anywhere in the body and particularly in this vulnerable area is highly unsurgical, and not to be tolerated. Far better to control bleeding for a few moments by digital pressure or by localized pressure with a stick sponge, or in this area by upward pressure of the finger inserted into the foramen of Winslow, and then to proceed with cautious clamping of the bleeding point as it is accurately revealed by the rapid removal of blood by suction. The initial brisk bleeding frequently gives way after only a few moments to a less terrifying loss of blood more easily controlled by slight pressure and by accurately placed clamps.

The cystic duct, having been identified and mobilized down to its junction with the common duct, is transected between clamps applied close to its termination, and the gallbladder is removed (Fig. 2b).

Immediately after removal of the gallbladder, the following steps performed by the operator with due observance of asepsis, bile being considered infected, should not be omitted: the gallbladder and cystic duct are laid open with scissors in order, first, to culture the bile for type and sensitivity of any organisms present, and, second, to inspect carefully the transected cystic duct. One must make absolutely certain that only the cystic duct has been divided and that no portion of the common duct has been removed with it. Also, the diameter of the cystic duct is carefully observed for comparison with the size of the gallstones within the gallbladder. Decision to open and explore the common duct may depend occasionally on whether it appears feasible that stones of a size found in the gallbladder could have migrated through the cystic duct into the common duct.

After the bile has been cultured and the caliber of the cystic duct examined, and when the decision to be content with a simple cholecystectomy has been made, the cystic duct is ligated close to the common duct with a long strand of No. 000 silk. One end of the

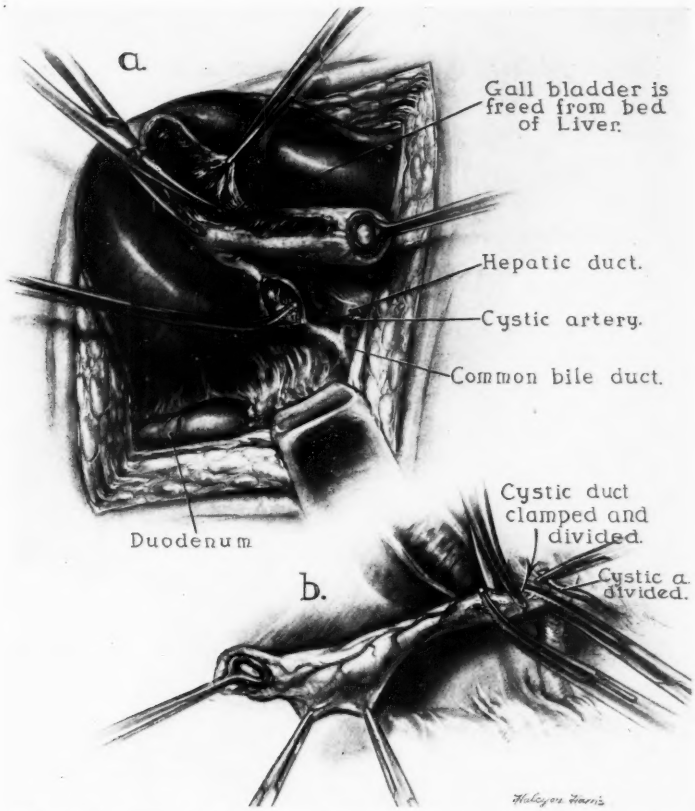

Fig. 2. To prevent injuries to normal or anomalous right hepatic arteries, and to identify without shadow of doubt the common and hepatic ducts, it is wise to mobilize the gallbladder from fundus to cystic duct. The latter is divided close to its junction with the common duct.

ligature is then threaded on a small curved, preferably French eye needle, and the duct is transfixed just *distal* to the ligature to avoid puncturing the duct where bile can escape (Fig. 3a). This transfixion is deemed necessary to prevent blowing off of the ligature should intraductal pressure subsequently become raised due to temporary edema of tissues at the ampulla produced by the trauma of palpating this area for stones. The cystic artery is ligated separately, preferably with silk or cotton. Usually this is the only vessel requiring ligation. Bleeding points in the gallbladder bed are seared with the Bovie unit, or ligated with fine catgut. The stumps of the ligated cystic duct and ligated cystic artery are then buried by approximating over them the previously incised peritoneum with several interrupted silk sutures (Fig. 3b). Covering the ligated cystic duct with peritoneum provides optimum conditions for healing. Experimentally, when an artery or cystic duct is ligated, the tissue included within the ligature dies, and permanent closure depends upon fibrous substitution of this necrosed tissue. Normally, the steps in this healing process are: (1) the accumulation of tissue fluid and exudate around the stump, (2) the deposition of fibrin in this exudate, and (3) the transformation of fibrin into fibrous

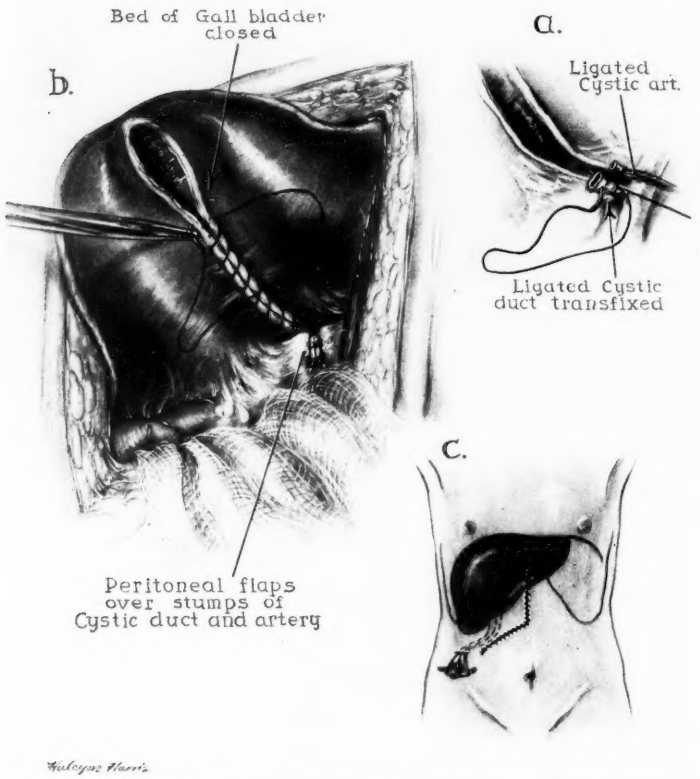

Fig. 3. a, The cystic duct is ligated *and* transfixed just beyond point of ligation. The cystic artery is ligated separately with silk followed by peritonealization of the area (b). b, The gallbladder bed is closed to block divided abnormal, usually invisible, branches of the biliary ducts when present, as they are capable of flooding the wound with bile. c, Two rubber cigarette drains, placed in the region of the gallbladder bed and foramen of Winslow, escape from the abdomen through a separate stab wound instead of through line of incision to prevent seepage of bile into and between wound layers.

tissue. If the initial tissue fluid is directed away from the site of ligation as, for example, by drains placed in close apposition, the deposition of fibrin and fibrous tissue is prevented or delayed, thereby increasing the hazard that the ligature may cut through the duct and cause massive bile drainage. Peritonealizing the ligated stump insures that the healing exudate is confined to the exact site where most needed, around the ligature.

The raw peritoneal edges of the bed of the gallbladder are approximated with a continuous catgut suture on an atraumatic needle (Fig. 3b). This closure controls bleeding and blocks the leakage of bile from liver cells inadvertently damaged in the mobilization of the gallbladder, but more important still, it effectively closes small, usually invisible, abnormal bile ducts which occasionally connect the intrahepatic biliary ducts with the gallbladder. Leakage from such divided ducts may explain the occasional copious but temporary discharge of bile following operation which cannot be attributed to reopening of

the cystic duct as the drainage of bile ceases so promptly. To provide for the escape to the outside of this occasional, unexpected, and unpredictable leakage of bile, it is imperative to introduce two small-sized latex rubber drains through a separate stab wound in the flank, both drains leading to the region of the gallbladder bed (Fig. 3c). *Under no circumstances should the drains be placed in contact with an unperitonealized ligated cystic duct.* This invites failure in healing and a reopening of the cystic duct. Also, these drains should not escape through the incisional wound, since the seepage of bile into the muscle layers increases the hazards of wound abscess and delay in healing.

Exploration of the common duct. If indications demand exploration of the common duct, the gallbladder is not immediately removed but is retained for traction and manipulation. A longitudinal incision, 1.5 to 2 centimeters long, is made medial and anterior to the cystic duct between traction sutures of silk. Careful note is made of the character of the first escaping bile—flecks of sandy debris betray the presence of a stone. Small flexible scoops are used to explore the duct, and we are not content until a rubber catheter, or a flexible probe, or a malleable scoop, has been introduced through the ampulla into the duodenum thus permitting palpation for stones along the entire common duct accurately located by the probe or catheter within it. Following exploration, drainage of the common duct is usually provided, but may be omitted if the opening into the duodenum easily admits a large catheter. The purpose of drainage is decompression of the duct which might become distended with bile through temporary closure of the ampulla by the edema incident to operative trauma. Rarely is a T tube used. Decompression through drainage is effectively accomplished by inserting a small catheter through the stump of the cystic duct (which may need to be dilated) or through a separate small stab incision in the duct, as reported by Reid. It is anchored in place by a tie of fine plain catgut around the cystic duct which *does not transfix* the catheter, thus avoiding interference with the early removal of the catheter

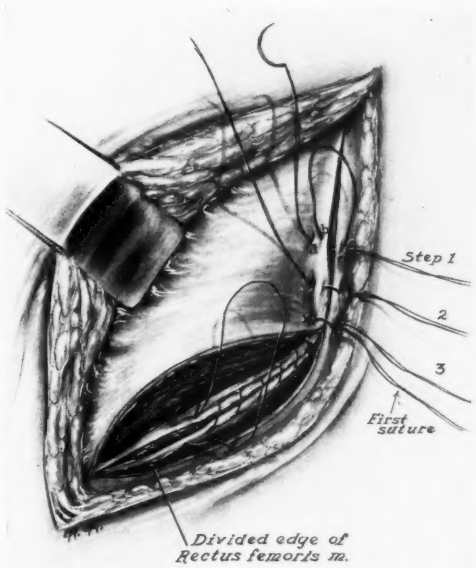

Fig. 4. The closure of a Kehr incision is critically important: points *x* and *y* at the caudad end of the midline incision are approximated first. Following closure of peritoneum and posterior sheath, the midline raphe and anterior sheath are approximated by interrupted end-on or vertical mattress sutures which insure accurate approximation of aponeurotic edge to aponeurotic edge—an important point in dependable healing.

when bile drainage ceases. The incision in the common duct is completely closed with a continuous fine plain catgut suture on an atraumatic needle, reinforced with interrupted silk sutures in the periductal tissues. Primary healing of this incision in the common duct should not be jeopardized by a foreign body such as a catheter or T tube passing through it. The catheter together with the drains to the gallbladder bed escapes through the separate stab wound in the flank. To avoid excessive or unnecessary syphonage of bile, the bottle containing the end of the rubber tubing connected with the catheter in the common duct is placed not more than 6 to 10 inches below the level of the common duct. On the third or fourth postoperative day the bottle is placed at a level slightly above that of the common duct. When bile flows readily into the duodenum and not into the bottle, the

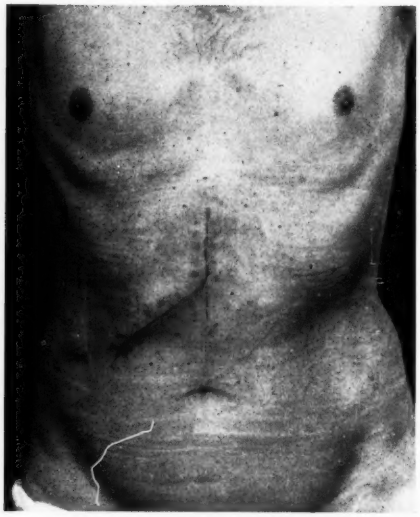

Fig. 5. Photograph of patient to illustrate landmarks and exact location of Kehr incision for cholecystectomy. Note site in flank where postoperative drains escaped from the abdomen. Operation July 11, 1951; photograph July 16, 1951.

catheter is withdrawn. This may be as early as the fourth day. If bile drainage is excessive or prolonged, choledochograms are made to detect or rule out overlooked stones.

Reconstruction of common duct. In operating for reconstruction of the common duct following operative injuries, the Kehr incision usually provides an adequate approach. If additional exposure is found necessary, the costal margin is further mobilized by division of the costal cartilage opposite the fifth intercostal space, as reported earlier (2).

Closure of the abdominal wound. The proper closure of the Kehr incision is critically important (Fig. 4). In preantibiotic days, the fear of possible infection and breakdown of this incision may have been responsible for the reluctance on the part of American surgeons to utilize this incision, since failure of healing would have produced a most deplorable type of ventral hernia. Closure of the Kehr incision entails the following steps (Fig. 4): (1) accurate approximation of point *x* to point *y* with a nonabsorbable suture applied as an end-on vertical figure-of-eight suture ("big bite, little bite, little bite, big bite"); (2) reapproximation of peritoneum and posterior sheath with a continuous chromic catgut suture; (3) reapproximation of the anterior sheath with interrupted nonabsorbable end-on figure-of-eight silk or cotton sutures; (4) reapproximation of the midline raphe by interrupted nonabsorbable end-on figure-of-eight sutures; (5) interrupted sutures in the skin complete the operation (Fig. 5). For nonabsorbable sutures, silk or cotton is ordinarily used, but No. 32 stainless steel wire is preferable if pus is encountered in the gallbladder. Also, they are not applied as *strangulating* sutures, but as *approximating* sutures. Contrary to almost universally held opinion, nonabsorbable sutures are particularly desirable should infection occur. In the presence of infection, catgut sutures are subject to early liquefaction and disintegration, thus increasing greatly the danger of wound disruption or of late herniation. Far better to be annoyed several weeks later by the necessity of removing a few silk sutures with the aid of local anesthesia and a crochet hook than to be embarrassed by early wound dehiscence or by the late development of an incisional hernia.

For closure of the midline raphe, the end-on nonabsorbable mattress suture is considered not only important but imperative. The two small inner bites *b* and *c* insure accurate junction of the two cut surfaces of the midline aponeurosis, and obviate the possible approximation of the aponeurotic edge of one side to fat on the other side, or the possible interposition of fat between the two cut edges, which might occur with the simple two bite suture. Also the far bites *a* and *d* provide a broader approximation of the midline aponeurosis with the added advantage of removing tension from the accurate junction of fascia edge to fascia edge provided by the near bites *b* and *c*. All these features improve the conditions under which healing of the midline raphe occurs, and provide greater insurance against accidental dehiscence.

Preoperative and postoperative care. When cholecystectomy is not an emergency procedure, certain precautions are imposed for a week to 10 days before operation and again after operation during the healing period. Obviously, the patient should be in a state of

optimum nutrition to endure successfully the ordeal of operation, as pointed out previously (1). Increasing evidence indicates that the all important adrenal and pituitary glands, with their high content of ascorbic acid, require liberal quantities of vitamin C to function properly under conditions of stress. Also, normal hepatic function is said to be dependent upon the adequate ingestion of vitamin B. Hence the prospective candidate for cholecystectomy is required for 10 days before operation and for variable periods after operation to take 1 or 2 multivitamin, multimineral capsules daily, and the juice of 2 oranges and 2 lemons daily. In the emergency cholecystectomy, ascorbic acid and vitamin B are provided in the parenteral fluids administered during and after operation.

The immediate postoperative care is concerned mainly with maintaining the activity of the patient in bed, by a 90 degree turn every hour, preferably through his own efforts, by exercising arms and legs frequently, and by deep breathing exercises every half hour when awake. Patients who have undergone laparotomy (in contradistinction to thoracotomy, or thyroidectomy or similar operations) are not required to get out of bed until the third or fourth day after operation. Early ambulation of a patient with a large abdominal incision is an ordeal of suffering that few surgeons would accept were they the victims. Moreover, the beneficial effects of the brief period of movement involved in 5 minutes of such ambulation are not to be compared with the benefits derived from the frequently repeated movements of hourly turning, of breathing deeply and of exercising of arms and legs every 30 minutes, activities imposed both night and day when the patient is awake.

The latex drains are usually removed on the third day, but removal is delayed for 3 to 4 days more if bile is found escaping along the drain at the first dressing.

Antibiotic therapy is administered beginning the day of operation (penicillin 400,000 units, streptomycin ½ gram, intramuscularly twice daily) but discontinued promptly if the bile culture is negative. If the culture is positive, antibiotics are administered for 5 days, or longer if conditions warrant.

SUMMARY

As a means of avoiding injuries to vital structures in the course of cholecystectomy, the Kehr incision first presented in 1913 is represented and re-advocated because of the directness of its approach and the resulting improved exposure of the common duct. The Kehr incision runs downward in the midline from the xiphisternum half way to the umbilicus, turns obliquely to parallel the midcostal margin and ends at the lateral edge of the right rectus muscle.

The steps in cholecystectomy are:

1. Careful identification of the cystic and common ducts.

2. Application of a clamp on the cystic duct to prevent migration of stones into the common duct during manipulation of the gallbladder.

3. Mobilization of the gallbladder from fundus to cystic duct to permit completely accurate identification of (a) the cystic and right hepatic arteries, (b) the cystic and common ducts.

4. Transection and transfixion ligation of cystic duct close to common duct.

5. Separate ligation of cystic artery.

6. Immediate opening of gallbladder (a) to obtain culture of bile, (b) to determine the internal diameter of cystic duct in relation to size of stones in gallbladder, (c) to make absolutely certain that no fragment of common duct is attached.

7. Peritonealization of the ligated cystic duct and artery to provide optimum conditions for healing.

8. Closure of the gallbladder bed by continuous catgut suture to control leakage of bile from divided invisible abnormal ducts between the intrahepatic biliary system and gallbladder when present.

9. Drainage of the operative area by drains placed in the region of gallbladder bed and foramen of Winslow which escape through a separate stab wound in the flank.

10. Closure of the incision by continuous chromic catgut in peritoneum and posterior sheath and by interrupted nonabsorbable end-on mattress sutures of silk, cotton, or stainless steel applied to the midline raphe and to the anterior sheath of the rectus muscle. Catgut

closure of these latter structures is contraindicated because of the increased hazard of early dehiscence or late herniation.

11. If exploration of the common duct is found necessary, drainage of the duct is effected by a small catheter introduced through the dilated cystic duct or through a separate stab incision in the duct, in order to permit complete closure of the exploratory incision in the duct.

12. In reconstructive procedures on the common duct, the Kehr incision usually suffices, but when better exposure is considered necessary, transection of the costal cartilage opposite the fifth intercostal space permits lateral and upward displacement of the costal margin and of the underlying liver, providing an excellent view of the subhepatic region.

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ANTIBIOTICS FOR SURGICAL INFECTIONS OF THE GASTROINTESTINAL TRACT

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OPTIMAL therapy of gastrointestinal tract infections of surgical interest requires more than familiarity with the drug employed, proper dosage, and preferred route of administration. The understanding of host-parasite relationships, antibiotic-parasite relationships, and antibiotic-host interactions is also requisite to intelligent management of these infections. For this reason the major portion of this presentation will be devoted to some of the criteria on which the current trends in therapy are based.

HOST-PARASITE RELATIONSHIPS

The gastrointestinal tract normally harbors a great many micro-organisms, the number and character varying with the segment concerned. In their normal habitat they may perform useful functions, including partial digestion of complex foodstuffs, synthesis of certain vitamins, and, by virtue of the antibiotic-like antagonisms of the normal flora, the suppression or elimination of ingested disease-producing micro-organisms.

This indigenous flora does not damage the host unless predisposing factors upset the functions which normally restrain them. Disturbance of the normal host-parasite relationships may occur under a variety of circumstances which include the following:

1. Intestinal obstruction. There is commonly ascent of the bacterial flora of the distal bowel proximally, as manifested by "fecal" vomiting. The presence of these micro-organisms in the upper gastrointestinal tract may contribute to the physiologic derangements associated with paralytic ileus.

2. Inflammation of the gallbladder, pancreas, or duodenum may result in bacterial invasion by fecal organisms.

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3. Increased migration of the normal intestinal flora or their metabolites through the gastrointestinal tract may injure the liver and promote hepatic necrosis. This is suggested by a number of experimental observations. For example, Gyorgi and Stokes, feeding rats a "necrogenic" experimental ration, were able to prevent or delay significantly the production of hepatic necrosis by mixing aureomycin with the diet. This antibiotic presumably exerted its beneficial effect by the suppression of intestinal bacteria and their toxic metabolites.[1] Death from liver hypoxia following injury or ligation of the hepatic artery has been shown by Markowitz and others to be prevented by the use of oral antibiotics, in experimental animals, and, possibly, in man. Several clinics have reported the recovery of some patients with hepatic coma following hepatitis or portacaval shunt procedures who were given adjunct aureomycin or terramycin therapy intravenously and by mouth.

4. Conditions which increase the permeability of the bowel wall such as uremia, especially when so severe as to be associated with mucosal damage and, possibly, shock. Frank and associates have demonstrated that death from profound hemorrhagic shock in dogs may be prevented or delayed if the animals are pretreated for several days with aureomycin or neomycin administered orally. Nelson and Noyes have found that the dog in hemorrhagic shock shows a significantly increased concentration of orally administered Clostridium botulinum toxin in the blood over control animals given identical amounts of the material. This suggests increased absorption of the toxin from the gastrointestinal tract or decreased removal from the blood stream in

[1] A crucial experiment on this point was described by T. D. Luckey of the Germ-free Laboratory, Notre Dame University, at the Symposium on Liver Disease at the New York Academy of Science, February 13, 1953. Preliminary trials show that Gyorgi's necrogenic diets do not produce liver damage in rats which, throughout their lives, are kept germ-free.

hemorrhagic shock. The clinical significance of these findings is not entirely clear, but suggests that there may be a relationship between metabolites of the normal gastrointestinal flora and the clinical course of deep hemorrhagic shock.

5. Conditions which damage the bowel wall. Inflammatory disease, vascular impairment, luminal obstruction, or injury to the intestine may lead to bacterial invasion of the bowel wall and peritoneal cavity. When coliform bacilli gain entrance to the blood stream, irreversible shock and death may result as is dramatically demonstrated by the unwitting transfusion of contaminated blood. Borden and Hall, of Minneapolis, and Pittman, of the National Institutes of Health, have reported 18 such fatalities.

The normal bacterial flora may interfere with the healing of the diseased or injured gastrointestinal tract, since healing of the bowel wall, as in other tissues, is optimal with an adequate blood supply and a sterile field.

6. Oral antibiotic therapy. This may allow preferential growth of certain micro-organisms such as monilia, staphylococcus aureus, pseudomonas, or proteus. These abnormal flora may result in or be associated with acute ulcerative or membranous colitis or proctitis.

7. Ingestion of large numbers of pathogenic organisms with the production of specific infections such as amebiasis, dysentery, or typhoid.

ANTIBIOTIC-PARASITE INTERACTIONS

The antimicrobial spectra of the different antibiotics currently available for groups of micro-organisms are shown in Figure 1. Bacitracin, neomycin, aureomycin, and terramycin exhibit nonspecific activity in the treatment of human amebic colitis. Aureomycin and terramycin are active in the treatment of Trichomonas vaginalis infection when they are applied topically, and terramycin by mouth is effective against enterobiasis (pinworm infestation).

Various strains of bacteria within a group or species show a marked variation in susceptibility to antibiotics. Our studies indicate the following trends in bacterial sensitivities: (a) Hemolytic streptococci are uniformly susceptible to all the antibiotics tested except streptomycin, neomycin, and polymyxin. (b) About 85 per cent of staphylococci are susceptible to erythromycin, carbomycin, and chloramphenicol, whereas only 50 to 60 per cent are susceptible to penicillin, streptomycin, and terramycin. In the past year the incidence of aureomycin and terramycin-resistant staphylococci has increased, and the incidence of penicillin-resistant staphylococci has declined. (c) Eighty per cent or more of Escherichia coli are susceptible to streptomycin and the "broad spectrum" antibiotics. (d) About 50 per cent of proteus species are susceptible to streptomycin, chloramphenicol, and neomycin. Our earlier studies indicated that many strains of proteus were susceptible to high concentrations of penicillin, and that a combination of penicillin, streptomycin, and sulfonamide was sometimes synergistic against these organisms (13). Recently Waisbren and Carr have observed an additive, as well as synergistic, effect when penicillin and chloramphenicol were combined and reported upon in 23 cases of proteus infection treated with these agents. (e) Ninety per cent of pseudomonas strains are susceptible to polymyxin and only 6 per cent to aureomycin and chloramphenicol, while about one-third of the strains are susceptible to streptomycin and terramycin.

Other studies have provided a clue to still another factor which bears on antibiotic therapy, namely cross-resistance. Thus, we found that penicillin-resistant staphylococci may be simultaneously resistant to aureomycin, terramycin, and chloramphenicol and vice versa (5). Cross-resistance of coliform bacilli may occur between aureomycin and terramycin (6). The clinical significance of cross-resistance is that, for certain species of organisms, the choice of antibiotics is not as wide as might appear on the surface.

DRUG-HOST INTERACTIONS

The molecules of drug which arrive in active form at the site of infection represent a small fraction of the much larger numbers which were present in the administered dose. Most of the absorbed antibiotic, when in contact with the body fluids, is rendered inactive by

binding. In general, the following working hypothesis may be employed: for every 100,000 units of penicillin administered, a blood level of 0.1 unit per milliliter may be anticipated. With streptomycin, the average blood level after injection is 2 micrograms for each 0.1 gram administered. Six hourly oral doses of the broad spectrum antibiotics of 0.25 to 0.5 gram yield blood levels of 2 to 8 micrograms per milliliter.

In many surgical infections of the gastrointestinal tract the oral route of administration of antibiotics is precluded. Oral therapy carries with it the risk of poor absorption or chemical irritation following major gastrointestinal resections. Rectally administered antibiotics in adults and children are poorly retained and poorly absorbed and this route is not reliable for obtaining therapeutic blood levels. Furthermore, the influence of circulatory collapse on antibiotic absorption should be recognized. Our studies (3) with dogs in acute hemorrhagic shock have shown that aureomycin, terramycin, and chloramphenicol given by mouth are poorly absorbed. On the other hand, circulatory collapse does not affect the measurable blood levels of intravenously administered antibiotics. Altemeier reported similar findings in the dog in traumatic shock. Whenever circulatory failure is likely, therefore, as in toxemic infections, following prolonged surgery, and in posttraumatic oligemia, the intravenous route seems the only reliable means of providing prompt therapeutic concentrations of antibiotics in the blood.

The biliary tract and pancreas warrant special consideration. Our studies on patients in whom simultaneous assays were made of antibiotic concentrations in the gallbladder and in the blood serum are presented in Table I.

It will be noted that considerable variation exists between the concentrations of the various antibiotics in the gallbladder bile and the blood serum. The appearance of these drugs in the biliary tract depends on the ability of the liver to excrete them and the patency of the cystic duct; when the cystic duct is obstructed or the common duct is blocked proximal to the cystic duct, the drugs do not appear in the gallbladder bile even

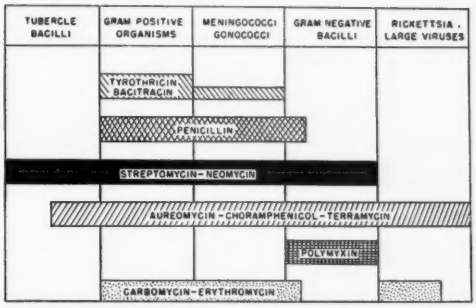

Fig. 1. Antimicrobial spectra of various antibiotics. Note that generic name of terramycin now is oxytelracycline.

though the drug is excreted from the liver In the unobstructed gallbladder, aureomycin, terramycin, bacitracin, erythromycin, and neomycin tend to appear in concentrations greater than in the blood, whereas penicillin, streptomycin, and chloramphenicol levels tend to approximate those in the blood serum. It is doubtful, in acute cholecystitis with cystic duct obstruction, whether antibiotics are able to reach the organ in concentrations sufficient to inhibit secondary invading organisms; however, it is reasonable to assume that they may afford protection against the infections and complications of acute cholecystitis. Studies on several patients with impairment of liver function have shown that excretion of these drugs in common duct bile is lessened. It is possible that therapeutic responses in infections complicating biliary tract obstruction are due at least in part to parenchymal distribution of the drug from the blood stream. In the absence of obstruction acute biliary tract infections are probably most effectively treated with antibiotics which are concentrated in the bile.

Excretion of antibiotics in the pancreatic secretions differs from that in the biliary tract. Drug excretion studies in 3 patients with pancreatic fistulas have been conducted at Walter Reed Army Medical Center (8). One fistula was the result of a gunshot wound and 2, of surgically marsupialized pseudocysts. Following intravenous administration, sodium sulfadiazine appeared promptly in the pancreatic juice in about the same concentration as in the blood; whereas, penicillin and strepto-

TABLE I.—COMPARISON OF ANTIBIOTIC LEVELS IN GALLBLADDER BILE AND BLOOD SERUM

Agent	Diagnosis	Blood level (mcg./ml. or u./ml.)	Gallbladder Bile level (mcg./ml. or u./ml.)
Bacitracin 25,000 units in 250 ml. 5 per cent dextrose 1 hour before surgery	1. Cholelithiasis	0.039	0.624
	2. Cholelithiasis	0.624	0.125
	3. Cholelithiasis	1.25	2.5
	4. Cholelithiasis	1.25	2.5
Penicillin 750,000 units in 250 ml. 5 per cent dextrose 1 hour before surgery	1. Cholelithiasis	5	5
	2. Cholelithiasis	10	10
	3. Cholelithiasis	20	2.5
	4. Cholelithiasis	10	2.5
Streptomycin 0.5 gm. in 250 ml. 5 per cent dextrose 1 hour before surgery	1. Cholelithiasis	5	5
	2. Cholelithiasis	0.6	2.4
	3. Cholelithiasis	16	8
	4. Acute cholecystitis with lithiasis	3.75	3.75
Neomycin 0.5 gm. in 250 ml. 5 per cent dextrose 1 hour before surgery	1. Cholelithiasis	0.6	2.5
	2. Mucous polyps	0.6	1.2
	3. Cholelithiasis	0.6	4.8
Aureomycin 0.5 gm. in 250 ml. 5 per cent dextrose 1 hour before surgery	1. Cholelithiasis	4	16
	2. Cholelithiasis	18	72
	3. Cholelithiasis	9	72
	4. Normal gallbladder	16	64
Chloramphenicol 0.5 gm. in 250 ml. 5 per cent dextrose 1 hour before surgery	1. Cholelithiasis	2	6
	2. Cholelithiasis	−2	−2
	3. Cholelithiasis	2	8
	4. Cholelithiasis	1.25	10
	5. Cholelithiasis (cystic duct obstruction)	0.8	−0.8
Terramycin 0.5 gm. in 250 ml. 5 per cent dextrose 1 hour before surgery	1. Cholelithiasis	4	8
	2. Cholelithiasis	8	64
	3. Cholelithiasis	8	64
	4. Cholelithiasis	18	72
	5. Cholelithiasis (cystic duct obstruction)	16	None
Polymyxin 100 mgm. in 250 ml. 5 per cent dextrose 1 hour before surgery	1. Cholelithiasis	4	4
	2. Cholelithiasis	1.6	9.6
	3. Cholelithiasis	0.6	9.6
	4. Cholelithiasis	4	—1
Erythromycin 400 mgm. per os	1. Cholelithiasis	0.156	10
	2. Cholelithiasis	0.156	1.25
	3. Cholelithiasis	1.25	20
	4. Cholelithiasis; subsiding acute cholecystitis	0.078	0.078
	5. Normal gallbladder	1.25	10

mycin appeared in lower concentrations. The other antibiotics (aureomycin, chloramphenicol, terramycin, and polymyxin) could not be detected in the pancreatic fluid. Inasmuch as the latter drugs are not inactivated by in vitro incubation in pancreatic juice, it seems likely that the pancreas does not excrete them.

The implications of these observations are that sulfadiazine, penicillin, and perhaps streptomycin should be used in the treatment of pancreatic fistula and in the management of patients subjected to surgical procedures involving marsupialization of a pancreatic cyst, pancreaticoenterostomy subsequent to subtotal resection of the pancreas, or internal drainage of a pancreatic cyst. Since penicillin and streptomycin both appear in the pancreatic secretion in low concentrations, the advantages of combined therapy should be borne in mind. It should be noted that experimentally produced pancreatitis is influenced favorably by orally, but not parenterally, administered aureomycin. It is probable that the beneficial effects attending therapy with this antibiotic result, at least in part, from suppression of the fecal flora rather than from pancreatic secretion of the drug.

THERAPEUTIC APPROACHES

The adjunctive use of antimicrobial agents against infections of the gastrointestinal tract may take one of three forms: (1) suppression of fecal flora during critical periods, (2) prevention of infection following surgery, and (3) treatment of established infections.

Suppression of fecal flora during critical periods. The aforementioned experimental observations of Gyorgi and Markowitz suggest that aureomycin or terramycin therapy is applicable to any condition which results in the multiplication of bacteria within the liver or in which liver hypoxia or liver damage may be aggravated by bacterial products. The favorable responses in some patients with hepatic coma are provocative and require further study.

The value of adjunct antibiotic therapy in acute cholecystitis is questionable. As already noted, in the presence of cystic duct obstruction, antibiotics cannot enter the distended gallbladder or its inflamed, thickened

wall. Their role in cholecystitis is, therefore, prevention or reduction in severity of the complications of the disease. The results of chloramphenicol therapy in patients who harbor typhoid bacilli in the gallbladder have been disappointing.

Bacterial invasion in acute pancreatitis and perforated peptic ulcer contributes heavily to the morbidity and mortality of these conditions. Sharp reductions in mortality since the antimicrobial agents have been added to the therapeutic armamentarium have been reported by Rhoads (pancreatitis) and Seeley (perforated peptic ulcer).

Acute intestinal obstruction with strangulation has been shown experimentally to result in bowel wall disintegration with passage of contents into the peritoneal cavity and blood stream despite intensive treatment for shock, hemorrhage, and electrolyte imbalance. Such animals treated with antibiotics show delay in destruction of the gut wall with prolonged survival time. It is, of course, hazardous to relate these observations to the corresponding condition in man. It would appear rational, however, to use antibiotics in conjunction with the relief of distention and treatment of shock, fluid, and electrolyte imbalance. Antibiotic therapy follows the same general principles used in peritonitis.

Prevention of infection following surgery. Peritonitis, the most frequent and most dreaded complication in surgery of the colon, can be prevented in most elective cases by careful technique at operation and by adequate preoperative preparation. Formerly, preparation for operations on the intestine consisted chiefly of starvation and elimination. As far as infection is concerned, these principles are still fundamentally sound. An empty bowel is usually at physiologic rest and its bacterial count is significantly reduced. Administration of enemas and mild catharsis, therefore, still remain important steps in preoperative preparation, while food by mouth is still held to a minimum. Parenteral alimentation for critical periods can take the place of the restricted food intake. Chemotherapeutic and antibiotic agents are used to supplement mechanical cleansing of the intestine. It should be stressed that these

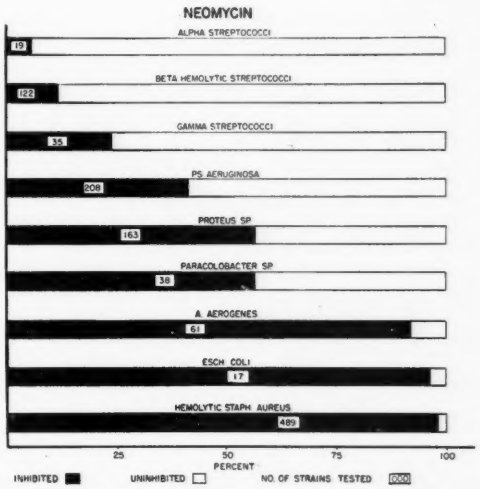

Fig. 2. In vitro neomycin sensitivity of bacteria from human sources (serial dilution technique). Organisms not inhibited by 20 micrograms per milliliter are considered neomycin resistant.

agents are most effective when they are put into a bowel cleansed of formed fecal material because these drugs must come in contact with the micro-organisms in order to kill or suppress them.

According to Poth, the ideal intestinal antiseptic should have the following properties: should have low toxicity for the patient, rapid (24 to 48 hours) activity, broad antimicrobial spectrum with minimal development of resistance, limited intestinal absorption, permit adequate food and fluid intake, assist in mechanical cleansing of the bowel without complete dehydration, be nonirritating, permit tissue healing, and be palatable. Poth believes that streptomycin, aureomycin, and terramycin are undesirable for use as intestinal antiseptics because of the rapid growing-out of resistant forms of bacteria with the former, and the gastrointestinal irritation and overgrowth of monilia attending the use of the latter two. In his broad experience, the most effective antibiotic is neomycin. The sensitivity of various bacteria to neomycin in vitro is presented in Figure 2. In most instances neomycin given orally completely eliminated susceptible bacteria from the intestinal tract within 24 hours. Following the elimination of

bacteria, yeasts grew out in large numbers. However, in his series of 350 cases, Poth did not encounter a single instance of yeast infection, nor did he encounter a single instance of bloody diarrhea or gastrointestinal tract ulceration following the administration of neomycin. Neomycin failed to inhibit the growth of Aerobacter aerogenes in approximately 10 per cent of the patients to whom the antibiotic was administered. This observation led him to combine neomycin with sulfathalidine. There does not appear to be any particular potentiation of either agent in the administration of this combination, but there seems to be a simple additive action when the two compounds are given together. In experimental animals, sulfathalidine and neomycin did not interfere with the normal processes of healing of the anastomosed bowel when given singly or in combination. The profound retardation of bacterial growth in the gastrointestinal tract attending intestinal antisepsis was not found to be accompanied by alteration of clotting, bleeding, or prothrombin times. Consequently, vitamin K and other supplements are not administered with these intestinal antiseptics unless the patient is found to have a prolonged prothrombin time or increased bleeding tendency, has been taking an inadequate diet, or is receiving a purified diet such as a protein hydrolysate. The recommended preoperative preparation is as follows: (1) low residue diet; (2) castor oil, 30 to 60 milliliters; (3) after the first liquid stool, administration of 1 gram of neomycin sulfate together with 1.5 grams sulfathalidine orally on a 4 hour schedule; (4) continuation of this dosage regimen for 2 or 3 days.

When given in this manner, the amount of neomycin absorbed into the systemic circulation is low, and Poth did not encounter a single instance of toxic reaction. Gastrointestinal surgery, performed by the open method of anastomosis, in neomycin-sulfathalidine-prepared patients did not result in a single instance of leakage from a suture linen or in the development of either peritonitis or fecal fistula.

The observations of Poth stimulated us to study the two following regimens for obtaining suppression of bacteria in the colon:

24 HOUR PREPARATION

0900	—Cathartic and cleansing enema	
1000		
1100		Total
1200	—Neomycin 1 gram	Neomycin 6 grams
1600	Sulfathalidine 1.5 grams	Sulfathalidine 9 grams
2000		
2400		
2100	—Tap water enemas until clear	

48 HOUR PREPARATION

0800	1st day	—Cathartic and cleansing enema	
0900			Total
1600	1st and 2nd days	—Neomycin 1 gram	Neomycin 6 grams
2300		Sulfathalidine 1.5 grams	Sulfathalidine 9 grams
2100	2nd day	—Tap water enemas until clear	

Two groups of patients were studied. Group I (normals) comprised those coming to surgery for minor anorectal conditions. Group II (abnormals) included patients with carcinoma, ulcerative colitis, or colostomy. The results in terms of swab cultures taken at the time of surgery are as follows:

24 hour preparation	No. cases	No. positive cultures	Percentage with positive cultures
Group I.....	26	5	19
Group II.....	18	12	67
48 hour preparation			
Group I.....	6	0	0
Group II.....	32	13	40

The organisms recovered at the time of culture included candida (monilia), streptococci, proteus, and pseudomonas against which neomycin has less activity than other fecal organisms (Fig. 2). These studies, while inconclusive when statistically analyzed, tend to confirm our studies with other antibiotics similarly tested in which we noted that maximal suppression of the fecal flora occurs 24 to 48 hours later in patients with colonic lesions than in normal individuals, and that the existence of ulcerative colitis, intestinal obstruction, intestinal fistulas, or perforations of the bowel associated with abscess formation interfere with the elimination of susceptible flora. The principal advantages of neomycin-sulfathalidine appear to be rapidity of action, low toxicity, and the fact that in the event of postoperative infection, the availability of less potentially toxic agents for systemic use does not deprive the surgeon of an important therapeutic agent for treating such complications.

Treatment of established infections. With good surgery and the advent of controlled colloid and electrolyte therapy, chemotherapeusis and antibiotic therapy, there has been a significant decrease in the case fatality rate of peritonitis. It is difficult to provide a definite guide as regards the antibiotic of choice for specific therapy on the basis of clinical experiences. Furthermore, it is not sufficient to choose an antibiotic for use in the mixed infection of peritonitis merely on the basis of its antibacterial spectrum, because penicillin, for instance, has little action upon Escherichia coli, the most predominant organism in most cases of peritonitis. Yet, penicillin, because of the role of gram-positive cocci in these infections, has been shown to be of definite value in reducing the mortality rate of peritonitis.

In an effort to evaluate the relative efficacy of the various antibiotic agents, experiments have been conducted at Walter Reed Army Medical Center (14) utilizing experimentally produced acute endogenous peritonitis as the test preparation. Rats and dogs have been used, since their normal bacterial flora approximates that of man. Peritonitis was induced by the operative perforation of the cecum followed by the intraperitoneal instillation (and the synergistic action) of gastric mucin. In test animals a single dose of antibiotic was given 4 hours after operation, at which time it was shown by sampling experiments that infection was present. Comparable doses of various antibiotics were administered intraperitoneally, intravenously, or by both routes simultaneously. No supportive therapy was administered. Control and test animals were followed for 96 to 144 hours, at which time all survivors were sacrificed. Only 12 per cent of untreated control rats survived. The use of any 1 of the 5 antibiotics used (penicillin, streptomycin, aureomycin, terramycin, and chloramphenicol) increased the survival rate over untreated controls.

In the data collected there is no statistically significant evidence that any of the antibiotics were superior to the others. It appears that terramycin, aureomycin, and a combination of penicillin and streptomycin are about equally effective, particularly so, if the latter is administered intraperitoneally in addition to the intravenous route of medication. Contrary to expectations, we have seen no untoward effects from the intraperitoneal instillation of dilute buffered terramycin solution, or solutions of penicillin and streptomycin.

The principal antibiotics employed in the prophylaxis and treatment of infections of the gastrointestinal tract may be summarized as follows: In perforated peptic ulcer and acute pancreatitis mixed therapy with penicillin (1,000,000 units) and streptomycin (1.0 gm.) given intravenously at 12 hour intervals may be employed for prophylaxis against infectious complications. Aureomycin or terramycin (1.0 gm.) intravenously, with or without concomitant oral administration of these drugs, appears useful in the adjunct therapy for infections of the biliary tract due to susceptible organisms. For intestinal obstruction or established acute secondary peritonitis, the aforementioned combination of penicillin and streptomycin, or aureomycin or terramycin employed singly may be used.

Terramycin, aureomycin, and chloramphenicol were effective in the treatment of large numbers of patients, in Korea, with Shigella dystenteriae. Four grams, in 3 doses, gave as satisfactory results as any other dosage regimens tested. Terramycin alone and in combination with other amebicides gave excellent initial responses and low relapse rates in amebic dysentery. A combination of aureomycin and chloroquine diphosphate was also effective. Chloramphenicol continues to be the only effective drug in typhoid fever. Polymyxin continues to be the only consistently effective drug in pseudomonas infections. Proteus infections pose a more difficult problem; a combination of a sulfonamide and chloramphenicol or streptomycin may be helpful. Erythromycin or carbomycin may be useful in the rare case of staphylococcal enteritis. The role of antibiotics in ileitis, ulcerative colitis, and lymphogranuloma venereum with rectal stricture is not clear. In the latter condition, use of antibiotics may lessen the degree of inflammation which is often associated with stricture.

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POLYVINYLPYRROLIDONE IN ACUTE TRAUMATIC AND HEMORRHAGIC SHOCK

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IN 1941 Schulz, Toennis (5, 6), and Orator published the first papers on the use of 2.5 per cent polyvinylpyrrolidone solutions[1] in physiologic saline in the treatment of shock due to trauma, hemorrhage, and burns. These reports dealt with the indications and dosages of PVP based on a wide experience as a substitute for whole blood and plasma in treating casualties both in the German Army and among the civilian population. Details of this experience were withheld until a publication by Hecht and Weese in 1943, who reported that thousands of clinical cases had been so treated. These authors emphasized that there is *no* substitute for whole blood in the more severe cases, but as a temporary measure in emergencies, as on the battlefield, PVP had found a very useful place. Schulz recommended serum or periston (PVP) during the first 12 to 24 hours in trauma and hemorrhage; and in burns he urged its use during the first 24 to 48 hours at a period when initial burn shock and hemoconcentration are greatest.

Experimental and clinical trials of polyvinylpyrrolidone have been uniformly successful. Repeated testing has failed to show any interference with blood typing and cross-matching, hematopoiesis, or with blood coagulation. PVP is nonantigenic and only rare and questionable side reactions have been noted.

Since PVP restores blood volume rapidly, is stable at a wide temperature range, is chemically uniform and metabolically inert, as well as relatively inexpensive and easily producible, it gives great promise of being an excellent supplement to our blood and plasma reserves.

We report here our experiences with PVP-macrose in the treatment of traumatic and hemorrhagic shock in 34 patients at Harlem Hospital, New York, with 1 added case of a highway accident.

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[1]PVP-macrose, used in this study, is 3.5 per cent polyvinylpyrrolidone in Ringer's solution. Polyvinylpyrrolidone, a white powder, soluble in water as a relatively nonviscous colloid was first synthesized chemically under certain pressures and temperatures from acetylene and formaldehyde. The molecular size of the material used clinically varies from 10,000 to 100,000. The K value of PVP-macrose supplied for this study is 31.

CLINICAL AND LABORATORY TESTS

In one group of 10 cases before, during, and after treatment, controlled observations were made on blood pressures, pulse rates, hematocrits, whole blood and plasma specific gravities, as well as on other vital signs. In addition, such liver function tests as cephalin flocculation, alkaline phosphatase, serum icterus index, van den Bergh, total serum protein, and albumin-globulin ratio determinations were done before, and at intervals after therapy. Urinary PVP excretion was followed in 1 case.

In a second series of 24 cases, shock was treated with PVP-macrose. Observations of blood pressure, pulse, and general clinical improvement were noted and recorded.

Summaries of the data in these two groups are presented in Tables I and II respectively.

A representative case is given in detail.

CASE V. C.P., Hospital No. 52-2115. The patient was admitted to the Harlem Hospital on February 3, 1952, after having been repeatedly stabbed in the body and extremities during an altercation.

On examination his pulse was imperceptible, blood pressure 0/0, respiration 22 per minute. The patient was a well developed, well nourished 38 year old colored male, conscious but ashen and unresponsive to questions and painful stimuli. There was evidence of excessive bleeding from a wound of the right brachial region anteriorly, just above the antecubital fossa. Another laceration 2 inches in length was found over the left anterior chest at the level of the third intercostal space just inside the anterior

TABLE I.—SUMMARY OF TRAUMATIC SHOCK CASES

Case No.	Nature of injury	PVP given	Change in clinical status	Change in hematocrit	Liver function studies	End result and comment
I JJ Age 47 Female 52-771	Cerebral concussion. Compound comminuted fracture of right tibia and fibula. Fracture of left ankle. Multiple lacerations. Shock	1,500 c.c. in 3½ hours	Prompt improvement in blood pressure and pulse, as well as other signs of shock	40 to 31.6% in 3 hrs. Rise to 33.3% at 24 hours	Negative	Discharged to home care on 76th hospital day without complication. Patient carried through operative procedure without use of blood or plasma
II NT Age 12 Male 52-1399	Severe cerebral concussion and laceration. Fracture of skull, left humerus, left radius, and left femur. Traumatic shock	1,000 c.c. in 4½ hours	Marked improvement in blood pressure, pulse, respiration, and general appearance of patient. Patient remained in coma with frequent convulsions for approximately 2 weeks	40.8 to 22.8% in 4½ hrs. Rise to 28.1% at 21 hours	Cephalin flocculation 3+ on 2nd day. Negative on 5th day and subsequently	Discharged on 46th hospital day much improved and fractures healing well
III HW Age 28 Male 52-1520	Severe basilar and right posterior parietal skull and mandibular fractures. Traumatic shock	1,000 c.c. in 2¼ hours	Marked improvement in blood pressure and pulse character	34.6 to 28.3% in 1 hour		Patient died 2¼ hours after admission. He was responding to treatment for shock, but died an asphyxial death due to aspiration of blood and mucus
IV LT Age 47 Male 52-1664	Penetrating stab wound of abdomen with laceration of liver. Traumatic and hemorrhagic shock	2,000 c.c. in 3 hours	Improvement in blood pressure, pulse, and other physical signs of shock	50 to 43.6% in 1 hour. Rise to 45% in 40 hours	Negative	Hospital course uncomplicated. Discharged in good condition on 16th day. Patient recovered from shock and was carried through a general anesthesia and laparotomy without the use of blood or plasma
V CP Age 38 Male 52-2115	Multiple lacerations of body and extremities with severe hemorrhage. Penetrating stab wound of left anterior chest	1,500 c.c. in 3 hours	Prompt and sustained improvement in blood pressure, pulse, and other vital signs	32.5 to 23.3% in 3 hours. 24.5% at 24 hours. 22.5% at 73 hours	Questionable positive cephalin flocculation on 15th hospital day	Discharged in good condition on 32nd hospital day. Patient was kept in hospital to observe the course of severe secondary anemia following loss of blood. No transfusion was given, but patient was improving steadily at time of discharge
VI BB Age 42 Male 52-3037	Laceration of temporal artery with severe hemorrhage. Hemorrhagic shock	2,500 c.c. in 3 hours	Immediate improvement in blood pressure with secondary fall with administration of 100 mgm. of demerol for sedation about 1¼ hours after starting treatment. Further improvement in pressure, pulse, and general condition of pt. upon further PVP administration	42.9 to 27.8% in 4¼ hours. Rise to 32.7% at 25¼ hours	Cephalin flocculation 3+ on day of admission	Discharged in good condition on 21st hospital day. Positive cephalin flocculation not attributable to PVP since initial blood was taken before administration of PVP and subsequent tests were improved and negative
VII LA Age 32 Male 52-3339	Penetrating stab wound of abdomen with laceration of liver. Traumatic shock and hemorrhagic shock	1,000 c.c. in 45 minutes	Prompt and sustained recovery from shock	40.9 to 34.1% in 1 hour. Rise to 35% at 6 hours	Negative	Anesthetic death during laparotomy 15 hours after admission. Response to shock treatment good. Death due to causes unrelated to PVP
VIII DA Age 45 Male 52-3670	Cerebral concussion. Compound fracture of right tibia and fibula. Fracture of right radius. Multiple contusions and lacerations of scalp and extremities. Traumatic and hemorrhagic shock	1,500 c.c. in 1¼ hours	Prompt and sustained recovery from shock	46.3 to 34.1% in 3 hours. Rise to 35.7% at 24 hours	Negative	Discharged on 97th hospital day in good condition
IX RG Age 38 Female 52-3871	Multiple penetrating stab wounds of the chest. Left hemopneumothorax. Hemorrhagic and traumatic shock	2,500 c.c. in 1 hour	Prompt improvement but not complete recovery from shock in time of administration of PVP. Blood pressure rose from 40/0 to 84/52 and the pulse which was initially imperceptible was 114 per minute and of fair quality	42.9 to 24% in 1 hour. Rise to 34.1% in 12 hours	Negative	Discharged in good condition on 14th hospital day. Patient was responding to shock treatment but because it was felt that she was having dangerous bleeding from a splenic injury, blood was given as soon as it was obtained
X JD Age 44 Male	Laceration of right forearm and left face with severe hemorrhage. Shock	1,200 c.c. in 2 hours	Prompt and sustained recovery from shock. Because of severe pallor and continued restlessness, though blood pressure was 100/60, 500 c.c. of blood were given after PVP. Pt. improved further after blood	35.2 to 24.4% in 2 hours. Rise to 27% at 24 hours	Negative	Discharged on 5th hospital day in good condition

axillary line. Moderate subcutaneous emphysema was felt about this wound with normal resonance to percussion and normal breath sounds over both lung fields. There was a small laceration of the right hand. The heart was not enlarged; sounds were weak and distant. The abdomen was flat and soft with no masses palpable. The remainder of the physical examination was normal.

Roentgenologic examination of the left chest following admission showed a fracture of the anterior aspect of the fourth rib. No evidence of pneumothorax was present following stabbing. There was obliteration of the right costophrenic angle.

PVP was started 1 hour after injury. Bleeding was controlled by clamps. The first 500 cubic centimeters of PVP was completed in 30 minutes. Blood pressure at that time was 70/40, pulse 80 per minute, quality weak. The patient was moving, and talking at random. The wounds were sutured and dressed. Within 1 hour the second unit of PVP was completed. Blood pressure at this time was 104/60, pulse 78 per minute, and of fair quality. The patient was more responsive and able to answer questions rationally. One and one-half hours after starting treatment the brachial wound had to be reopened because of recurrent bleeding. Suture ligatures were placed to control the bleeding and the wound was reclosed. The blood loss at this time was approximately 200 cubic centimeters. At the end of the procedure, the radial pulse was palpable. The blood pressure was 110/78, pulse 68 per minute, and of fair quality, 3 hours after starting treatment. The third unit of PVP was completed at this time and the infusion stopped. The patient complained of thirst during the first hospital day and drank approximately 2,500 cubic centimeters of fluids. Urine output for the first 24 hours was about 1,000 cubic centimeters. After recovery from shock the mucous membranes and nail beds remained very pale. Temperature, pulse, and respirations remained within normal limits, though there was some increase in the subcutaneous emphysema in the region of the left anterior axillary fold.

Four days after admission, blood pressure was 100/60, pulse 88 per minute, and temperature 98.9 degrees F.; respirations were 18 per minute. Hemoglobin was 42 per cent. No subcutaneous emphysema was present over the left chest. Six days after admission, the blood pressure was 128/80, pulse 84 per minute and of good quality. The patient now was out of bed and ambulatory. There was no dyspnea, but a feeling of mild pain in the left chest in the region of the rib fracture.

The blood picture and hemodynamic changes during the first 121 hours in the hospital are shown in Table III. The patient's course was uncomplicated. He received no blood or plasma. He developed a voracious appetite and consumed quantities of food on the ward. He was discharged on the thirty-second hospital day in good condition with the anemia steadily improving. Laboratory tests up to discharge were essentially negative other than a severe secondary anemia and hypoproteinemia.

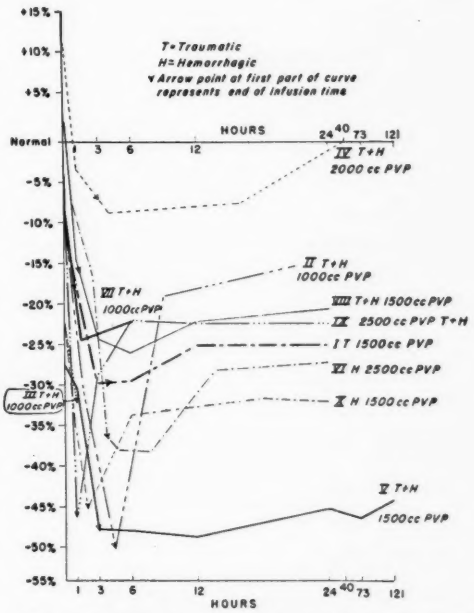

Fig. 1. Per cent changes in hematocrit in patients with traumatic and hemorrhagic shock receiving PVP infusions.

DISCUSSION

Of the 10 patients studied in detail 2 were females, 8 were males, with ages ranging from 12 to 47 years. The types of injuries varied from one of pure hemorrhagic shock following laceration of the temporal artery (Case VI, Table I) to severe multiple wounds. Two of the patients had penetrating wounds of the chest in addition to other injuries (Cases V and IX), and 2 had penetrating wounds of the abdomen with lacerations of the liver (Cases IV and VII). Four had cerebral concussions complicating multiple fractures of skull or extremities (Cases I, II, III, VIII). All were in moderate to profound shock on admission.

PVP-macrose in volumes from 1,000 to 2,500 cubic centimeters (2 to 5 bottles) as infused within periods varying from 45 minutes to 4½ hours. Concomitantly surgical care was carried out in the usual manner. Two of these patients, who recovered from shock, were carried successfully through operative procedures with infusions of PVP as the *only* intravenous fluid used (Cases I and IV).

TABLE II.—CASES OF TRAUMATIC AND HEMORRHAGIC SHOCK TREATED WITH PVP

Case No.	Nature of injury Date	General condition before and after PVP	Blood pressure Before After	Pulse Before After	Reaction Result
1 GB Age 50 Male *	Laceration of scalp 1-19-52	Mild shock secondary to hemorrhage Improved after 1,000 c.c. of PVP	80/40 110/80	118 100	None Improved Discharged
2 JB Age 36 Male *	Stab wound of chest, nonpenetrating 3-29-52	Shock secondary to hemorrhage and trauma Improved after 500 c.c. of PVP	80/50 100/60	120 108	None Improved Discharged
3 MC Age 23 Female *	Lacerations of right breast and left posterior chest 4-12-52	Moderate shock secondary to hemorrhage and trauma Improved after 500 c.c. of PVP	70/30 108/60	118 104	None Improved Discharged
4 RE Age ? Female *	Laceration of left wrist 3-18-52	Mild shock secondary to hemorrhage Improved after 500 c.c. of PVP	78/50 100/80	100 96	None Improved Discharged
5 BE Age 25 Female 52-7592	Stab wound of chest, multiple lacerations 4-29-52	Moderate shock secondary to trauma and hemorrhage Improved after 500 c.c. of PVP	80/40 120/80	120 100	None Improved Discharged
6 AF Age 26 Male *	Laceration of scalp 3-23-52	Mild shock secondary to hemorrhage Improved after 500 c.c. of PVP	88/40 100/80	124 108	None Improved Discharged
7 SF Age 44 Male *	Laceration of right chest, abdomen, left hand, and chin 4-18-52	Moderate shock secondary to trauma and hemorrhage Improved after 1,000 c.c. of PVP	76/50 108/78	118 104	None Improved Discharged
8 AG Age 48 Male 52-9334	Penetrating gunshot wound of abdomen with injury to bladder and external iliac vein, and fracture of pelvis 5-30-52	Severe shock secondary to trauma and hemorrhage Improved after 1,000 c.c. of PVP. Immediate surgical exploration with use of 1,000 c.c. of whole blood	70/0 100/60	124 110	None Improved Discharged
9 NG Age 32 Male *	Laceration of right ear 5-2-52	Mild shock secondary to trauma and hemorrhage Improved after 500 c.c. of PVP	80/30 100/60	126 112	None Improved Discharged
10 JG Age 31 Male *	Laceration of scalp 2-12-52	Mild shock secondary to hemorrhage Improved after 500 c.c. of PVP	90/60 120/70	114 96	None Improved Discharged
11 WE Age 53 Male *	Laceration of scalp 4-8-52	Moderate shock secondary to hemorrhage Improved after 1,000 c.c. of PVP	78/30 114/80	118 88	None Improved Discharged
12 LM Age 43 Female *	Stab wound of chest, nonpenetrating, and multiple lacerations 4-26-52	Mild shock secondary to hemorrhage and trauma Improved after 500 c.c. of PVP	80/36 120/74	122 92	None Improved Discharged
13 GI Age 50 Female 52-9326	Cerebral concussion. Fracture of right femur. Bilateral fractures of both pubic rami 5-29-52	Severe shock secondary to trauma and hemorrhage Improved after 1,000 c.c. PVP; 1,000 c.c. of whole blood followed	40/0 118/76	122 88	None Improved Still in hospital
14 SJ Age 19 Male 52-10683	Stab wound of the heart 6-20-52	Severe shock secondary to trauma and cardiac tamponade Improved after 500 c.c. PVP; 500 c.c. of plasma and 500 c.c. of whole blood followed. Immediate thoracotomy with repair of wound of right ventricle	60/0 120/78	Imperceptible 88 (at end of operation)	None Improved with recovery Discharged

*Patients not admitted to the hospital were treated on the accident ward and referred to the out patient clinic for follow-up.

TABLE II.—CASES OF TRAUMATIC AND HEMORRHAGIC SHOCK TREATED WITH PVP—CONTINUED

Case No.	Nature of injury Date	General condition before and after PVP	Blood pressure Before After	Pulse Before After	Reaction Result
15 WJ Age 33 Male *	Laceration of neck 1-18-52	Moderate shock secondary to hemorrhage Improved after 1,000 c.c. of PVP	70/0 100/80	128 96	None Improved Discharged
16 EM Age 34 Female *	Laceration of buttocks 2-18-52	Mild shock secondary to trauma and hemorrhage Infusion after 500 c.c. of PVP	90/40 120/70	118 96	None Improved Discharged
17 JQ Age 44 Male *	Laceration of neck and left ear 2-13-52	Mild shock secondary to trauma and hemorrhage Improved after 500 c.c. of PVP	88/44 100/80	112 88	None Improved Discharged
18 HR Age 34 Male 52-7827	Cerebral concussion and fracture of acetabulum 5-3-52	Moderate shock secondary to trauma Improved after 1,000 c.c. PVP	80/50 130/80	110 100	None Improved Discharged
19 SS Age 36 Male 52-9695	Cerebral concussion. Compound fracture of left tibia and fibula 6-5-52	Moderate shock secondary to trauma and hemorrhage Improved after 1,000 c.c. of PVP	78/40 128/78	108 100	None Improved Discharged
20 CT Age 69 Female *	Stab wound of back. Laceration of scalp 2-4-52	Moderate shock secondary to trauma and hemorrhage Improved after 500 c.c. of PVP	90/44 110/70	118 94	None Improved Discharged
21 LT Age 26 Male 52-9383	Stab wound of pericardium 5-31-52	Marked shock secondary to trauma and hemorrhage Improved following 1,000 c.c. of PVP; 500 c.c. of plasma and 500 c.c. of whole blood followed. Immediate thoracetomy with exploration of the wound	60/40 110/60	100 112	None Patient died on 4th postoperative day from acute fulminating empyema, pericarditis and heart failure
22 AT Age 30 Male *	Laceration of right thigh 2-26-52	Mild shock secondary to trauma and hemorrhage Improved following 500 c.c. of PVP	98/80 118/80	108 92	None Improved Discharged
23 CT Age 26 Male *	Laceration of face, neck, and back 2-2-52	Mild shock secondary to trauma and hemorrhage Improved after 1,000 c.c. of PVP	80/40 110/70	108 88	None Improved Discharged
24 VU Age 37 Female 52-10114	Cerebral concussion. Fracture of body of L-2. Fracture of left talus 6-11-52	Severe shock secondary to trauma. Improved after 1,000 c.c. of PVP	0/0 110/80	Imperceptible 80	None Improved Discharged

*Patients not admitted to the hospital were treated on the accident ward and referred to the out patient clinic for follow-up.

In all, there was prompt and sustained improvement from shock. In the 2 cases in which death occurred, neither was attributable to failure of antishock therapy. One of these patients who had a severe head injury with brain laceration aspirated blood and mucus and drowned (Case III). The other died as a result of the anesthetic (Case VII).

In addition to the clinical improvement, significant decreases occurred in the hematocrits, with slow secondary rises after cessation of infusions. These changes are depicted in Figure 1. The peripheral blood and plasma specific gravities showed a similar hemodilution. After recovery (in several cases) from hemorrhagic shock, blood was purposely withheld to observe the course of these patients who were not given transfusions (Cases I, IV, V, and VI). No complications occurred. The deficiencies in hemoglobin and plasma proteins were rapidly restored. Liver function studies were questionably positive in

TABLE III.—BLOOD PICTURE AND HEMODYNAMIC CHANGES IN CASE V

	Before PVP	Period after administration of PVP						
		1 Hour	3 Hours	6 Hours	12 Hours	24 Hours	73 Hours	121 Hours
Hemoglobin, per cent				42		44	42	44
Hematocrit	32.5	31.3	23.3	23.3	22.9	24.5	22.5	25
Plasma protein, gms. per cent						Alb. 3.4 Glob. 1.9 Total 5.3		
Plasma, sp. gr.	1.0211	1.0202	1.0200	1.0192	1.0197	1.0214	1.0226	1.0244
Whole blood, sp. gr. Capillary	1.0423	1.0371	1.0352	1.0348	1.0338	1.0394	1.0388	1.0409
Venous	1.0392	1.0354	1.0342	1.0340	1.0332	1.0391	1.0383	1.0407
Blood pressure	o/o	104/60	108/68	100/60	128/70	140/100	100/60	128/80
Pulse	Imperceptible	78	70	88	100	100	88	84

only 2 cases (Cases II and V). No sensitivity reactions, either immediate or delayed, were observed.

In 1 patient, urinary excretion of PVP was measured. Forty-seven per cent of the infused PVP was accounted for in the first 3 hours and an additional 15 per cent in the second 3 hour period, making a total of 62 per cent excretion in the first 6 hours. These data agree with those of Fine (3) and our excretion studies with tagged C14 PVP.

The second group studied included 24 patients in shock. Sixteen responded so well to PVP infusions and surgical treatment in the accident ward that they were discharged to the outpatient clinic and only 8 required admission. Of these 1 died 4 days after operation (Case 21, Table II).

Our experience in this series is similar to the first; namely, in all instances, prompt clinical improvement followed the intravenous PVP with a rapid rise in blood pressure, a slowing of the pulse, an increase in pulse pressure, and an improvement in vital signs.

In conclusion, we are reporting an automobile accident in which a patient lapsed into profound shock as a result of severe multiple injuries of the pelvis and both lower extremities.[1] While awaiting the arrival of the ambulance he received 1,000 cubic centimeters of PVP-macrose within 20 minutes. The patient's condition showed steady improvement. On arrival at the Methodist Hospital, Brooklyn, 1 hour later, he was given 500 cubic centimeters of plasma and taken to the operating room where an emergency amputation of the right lower leg was rapidly carried out. He received 2,000 cubic centimeters of group A Rh positive blood in the theater.

No crush syndrome developed. He secreted 1,000 cubic centimeters of urine during the first day after operation. His subsequent course has been entirely satisfactory.

[1]Three compound comminuted fractures (right tibia and fibula, left femur); compound dislocation of head of left femur, and 5 other dislocations (both knees, ankles, and symphysis pubis); simple fracture of right femur, middle third; fracture of left tibia and fibula; multiple lacerations with appreciable blood loss.

CONCLUSIONS

1. PVP-macrose has been used successfully as emergency treatment in 35 cases of traumatic and hemorrhagic shock of all degrees of severity associated with head injuries, multiple fractures, and penetrating wounds of both chest and abdomen. Some of these patients required blood and plasma subsequently.

2. The clinical responses were judged to be good in all instances as evidenced by prompt rises in blood pressure, slowing of the pulse, increase in pulse pressure, improvement in vital signs, decrease in the venous hematocrit and plasma proteins, as well as dilution of the capillary blood as observed in the decline of the specific gravity of peripheral blood.

3. The urinary output was satisfactory in all.

4. No sensitivity reactions either immediate or delayed were observed. There were no toxic reactions.

5. No interference with blood typing, coagulability, or depression of hematopoiesis occurred.

6. Although PVP is slightly hyperviscous, it flowed readily in the near-freezing weather, when injected at the scene of the highway accident.

7. Our results fully substantiate previous reported experiences and indicate that polyvinylpyrrolidone is a very valuable adjunct in the emergency treatment of both traumatic and hemorrhagic shock.

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EDITORIALS

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SEPTEMBER, 1953

THE PALLIATIVE COLOSTOMY

THE SURGEON often must decide at the operating table whether or not to perform palliative colostomy. This dilemma has faced, and will face, not only those particularly interested in surgery of the colon and rectum but also those who do general abdominal surgery.

Even to the surgeon, whose special field of endeavor has equipped him with knowledge gained by personal experience and by familiarity with competent writings, the decision is not at all times an easy one. Although he usually has had time before the operation to evaluate many factors that may be present, when the abdomen is open the weight of additional evidence disclosed by exploration and examination of the pathologic lesion may well create a fine balance for and against colostomy.

The following factors carry great weight in evaluation of the situation: (1) obstruction, its degree and cause; (2) local fixation, malignant, not inflammatory; (3) distant metastasis, its extent; (4) location of primary malignant lesion; (5) ulceration and bleeding; (6) the grade of malignancy; (7) the age of the patient; (8) the social, intellectual, and economic status of the patient; and (9) the prognosis for life after palliative colostomy.

Obstruction varies in degree. The actual size of the lumen of the bowel, as it is encroached on by a combination of malignant and inflammatory growth, has been overemphasized as an indication for colostomy. A lumen 1 centimeter in diameter is compatible with life if obstruction to fecal or foreign material above it can be obviated. Not infrequently, it is most surprising to observe a high degree of obstruction and a low degree of secondary effect on the portion of bowel proximal to it. Serious and sudden obstruction can take place under such circumstances, such as following the ill-advised administration of barium by mouth. In other words, obstruction caused by a small lumen alone is not often an indication for palliative colostomy if careful and considerate measures are carried out to avoid obstruction secondary to that produced by the narrowing. Such measures include the giving of oil enemas when necessary, administration of mineral oil by mouth in doses adjusted to individual needs, and use of a low residue diet, to be followed finally by a nonresidue diet when indicated.

Local fixation is mentioned because it is not a simple matter to determine its degree and, in attempting to evaluate the resectability of any growth, to decide whether fixation is caused by local extension of the malignant process or by secondary inflammation.

Distant metastasis to lymph nodes along the aorta, and to the peritoneum, omentum, and liver may be so extensive as to permit only exploration and closure. The influence

of metastasis may vary widely. A metastatic growth in the liver may be single and of a size and location to permit its resection, in which case its resection is surgically justifiable in addition to extirpation of the bowel lesion. In those instances in which a nonresectable primary growth is present, together with metastasis to the liver, it is best to avoid palliative colostomy and to resort to nonsurgical and dietary palliation. The same may be said of cases in which there is a small primary growth with extensive spread to the liver.

The location of the primary malignant lesion is important because, if mere palliation is possible, the location together with other factors will determine what type of palliative technical procedure can be undertaken to avoid colostomy. From a surgical standpoint, if degree of metastasis does not make extirpation of a resectable growth out of the question and if other factors are favorable, it is wise to remove the primary feeder growth. When such a resectable malignant growth is in the rectum, palliative, single-barrel sigmoid colostomy occasionally may be indicated. However, it is surgically sound and in the best interest of the patient to avoid palliative colostomy and to perform palliative anterior resection and anastomosis whenever and wherever possible.

There are times when, because of ulceration, fixation, and hemorrhage, in the presence of extensive lesions of the rectum, palliative defunctionalizing colostomy is essential. Following such a procedure, local radium and roentgenotherapy are beneficial in some cases but, at best, life under such conditions is miserable for the patient and for those who come in contact with him.

With increased knowledge pertaining to the effect of the grade of malignancy on prognosis, it is safe to state that the higher the grade, the more reluctant one should be to perform palliative colostomy in the debatable case.

The age of the patient is an important influence. Despite the fact that one may feel hesitant, simply because of age, to perform palliative colostomy in cases of older patients in which proved, fixed, low grade malignant rectal lesions are present, there have been several instances in which such a procedure, followed by radium and roentgenotherapy, has resulted in a prolonged and relatively comfortable existence. On the other hand, it is well known that youth is a prognostic disadvantage in malignant disease.

Although one must approach the influences of the patient's social, intellectual, and economic status charitably and seemingly without discrimination when considering palliative colostomy, these conditions nevertheless are of great importance both to the patient and to the family on whom the burden of care must fall. They present problems that must be solved with wisdom and great consideration in each individual case.

Finally, in a review of cases in which palliative colostomy had been performed, the duration of life was found to have been only a little more than a month longer than it had been in cases in which such a procedure, and also resection, had been avoided. For this reason, with due consideration for the welfare of the patient concerned, and with recognition of other measures that can be carried out in his interest and for his comfort, a well-based opinion is that colostomy as a palliative procedure should be avoided whenever possible.

CHARLES W. MAYO.

STANDARDIZATION OF HOSPITAL SUPPLIES AND NOMENCLATURE

THE SHORTAGE of registered nurses is acute and it is apt to become increasingly severe. In 1951 about one-half of all the nursing care which was

performed in nonfederal, general hospitals was carried out by practical nurses, nurses aides, and the like.[1]

The facilities for the training of these "aides" and practical nurses have not been able to keep up with the demand. It is obvious that their numbers must be increased several fold in order to give adequate care to the sick.

One of the difficulties in training these "helpers" is that the instruments and equipment, such as dressings, with which they must become familiar are still named and made to the specifications set up by a large number of surgical and medical individualists who often considered that they could not attain proper recognition unless either their instruments, dressings, salves, or diets were named after them. As an example, in the catalogue of surgical supplies printed by Jetter and Scherer Company of Tuttlingen, South Germany, in the late 1920's, there were over 1,800 different types of knives, blades, and handles listed in one or another combination. This has been corrected in part as a result of the development of the detachable blade scalpel. One of the leading manufacturers suggests that a standardization of needles would require 165 different sizes or lengths of needles. However, they list 217 "special needles" in addition to the "standard needles" which they carry in stock. Actually, they offer 38 different needles for blood transfusions alone.

The confusion in diets, special feeding formulas or their variants, individually named in each hospital, adds to the difficulty and means that any transfer of helpers from one hospital to another will require a great deal of training simply to learn to speak the local language. The situation in regard to dressings is almost as bad.

[1]Facts About Nursing. American Nurses Association; 1952, p. 17.

The Hospital Standardization Movement of the American College of Surgeons was instituted about 1918.[2] The Commodity Standards Division of the Office of Industry and Commerce, U. S. Department of Commerce, has set up simplified practice recommendations and commercial standards which have been initiated by the American Hospital Associates Committee on Purchasing, Simplification and Standardization.[3] These recommendations cover a large number of items, being concerned particularly with stock sizes and varieties of specific commodities that are in general production and use at the present time.

The Bureau of Hospital Supplies and Equipment, New York, New York, The American Standards Association, The American Surgical Trade Association, and the American Society for Testing Materials are also actively helping the American Hospital Association in their effort to simplify and standardize materials which are used by hospitals.

It seems obvious that the medical profession should take an active hand in furthering these efforts which are almost unknown to most of us. The financial advantage of such an effort is easily visualized. Even more important would be the possibility of training auxiliary help so that they can more easily and quickly help the nursing profession provide adequate care for the sick.

An effective method for implementing such standardization of equipment and nomenclature would be by the united effort of the hospitals which now have approved residency programs. When we consider that there are now 1,388 approved residencies with approximately 4,000 men in training in general surgery alone, it is easy to see that if such a pro-

[2]Hospitals. April 1937, p. 55.
[3]Hospitals. June 1953, vol. 27, part 2, p. 401.

gram were adopted in these hospitals alone, it would soon be felt in every hospital in the country.

The American College of Surgeons, the American Medical Association, and the various societies which appoint the members of the various certifying boards should join with the American Hospital Association and the other groups listed in attempting to bring about a standardization of nomenclature as well as hospital supplies and equipment.

R. K. GILCHRIST.

THE SURGEON AT WORK

ANAL DELIVERY OF RECTAL TUMORS

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THERE are three distinct approaches to the disposition of certain rectal tumors of benign origin, viz., by delivery to actual grasp outside the anal canal, by diagnostic and therapeutic measures conducted through a proctoscope, or by abdominal surgery.

The first of these, delivery outside the anal canal, holds exclusive and undebatable advantages within its indications. Unfortunately, however, there are technical difficulties of accomplishment which are realized and fully appreciated only on actual trial. These have considerably restricted the potential field of usefulness and, as corollary, their surmounting would effect substantial clinical gains. In spite of these considerations we fail to find anywhere in the literature suggestions of adequate technique or for that matter of *any* technique. Literally, everyone has been left to flounder for himself.

We do not have in mind here the problems of carcinoma but of those polypoid and sessile tumors which are either benign or of uncertain character. These latter frequently exhibit deviations which are impossible to translate into unequivocal clinical terms of prognosis and therefore of assuredly adequate surgery. Thus there is frequently at issue problems of both interpretive pathology and surgical technique which combine to enhance the uncertainties and attendant responsibilities materially.

In this situation of dual aspect the anal delivery of a tumor in question may well prove the critical factor both in the determination of proper decisions and of their successful execution. On the pathologic side the gross characteristics, as ascertained by palpation, may transcend all else in diagnostic importance, while in addition a reliable microscopic report is far more assured by the exactitude of the site chosen for biopsy in comparison with working through a proctoscope. Lahey (1) recently epitomized the practical aspect of this pathologic phase when he stated: "In the broad base polyps that can be palpated I would much rather make the decision of the danger of malignancy by the feeling of induration in the base than I would by examining bits of polypoid portion of the polyp." In this and other discussions it has been overlooked that such a dictum obviously derives meaning only in proportion as polyps *can be* palpated. The deceiving "bits" mentioned are of course the contingencies and liabilities of the alternative proctoscopic approach.

On the surgical side too, success may hinge on the ability to deliver the lesion into the surgeon's grasp. Failure of this intra-anal approach inexorably commits one to less desirable alternatives. If the lesion is actually benign the commitment is to more difficult and dangerous approach through the proctoscope. If, by reason of the delinquencies mentioned, the lesion is mistakenly diagnosed as benign the commitment is also to proctoscopic approach which, in such instances, is not only more dangerous but inevitably and tragically inadequate.

No amount of sophistry can gainsay the comparative limitations of both diagnostic and therapeutic approach to rectal lesions through a proctoscope. Mention has been made of the inadequacies and deceptions of diagnosis implicated, while therapeutic measures go hand in hand with the inherent and constant dangers of perforation and hemorrhage. It is not necessary to reiterate here the fateful reality of these limitations and dangers. There can be no doubt that experience and skill are valuable assets in working through a proctoscope but the increased obstacles are beyond debate. And it must not be forgotten that the prohibitions, restrictions, and hazards which might be avoided by anal delivery become inexorably translated into final terms of morbidity and mortality.

The comparative magnitude of abdominal approach is obvious. Its avoidable performance would certainly constitute a gross disservice to the patient. On the other hand the ultimate catastrophes in choosing the less formidable alternatives on the basis of mistaken diagnosis are equally obvious. We venture it as incontestable that induration in the base of a sessile polyp, as determined by palpation, indicates radical abdominal surgery. Thus one might go far and tragically astray on the sole basis of negative and

misleading biopsies such as are likely when obtained through a proctoscope. Such mistakes are avoided by the satisfactory palpation and accurately revealing biopsy afforded by anal delivery.

One of the most provocative situations confronting the proctologic surgeon is that presented when tumors under discussion are situated approximately at the level of an examining forefinger. The circumstance may prove most beguiling until one discovers that touching the tumor and delivering it to serviceable grasp outside the anus are two far different things. Even lesions below this level may give unexpected trouble in delivery. Lesions appreciably higher are at once outside the field of any choice. It is particularly at the level mentioned that facility in the anal delivery of tumors becomes a critical factor in achieving maximum success. Failure at once consigns the patient to a less favored group.

This procedure is more difficult of accomplishment than is usually envisioned before attempted. Lack of an established plan, the inadequacy of instruments available, friability of tissues involved, and the constrictive obstacle of the anal canal are contributory to both anticipated and unanticipated difficulties. A too common experience is that, after repeated unsuccessful attempts at such delivery, bleeding ensues to obscure the field, and a disconcerted surgeon admits defeat.

Among the methods usually tried is that of "inching" the tumor down by successive grasps of two alternating forceps. But there is invariably found to be surprising elusiveness in even seizing the tumor. Granting success with this preliminary, additional adverse factors then come into play with any downward traction for delivery. Increasing tension of the bowel wall and its rapidly widening cone shaped mass combine to resist dragging through the narrowing channel of the anal canal. Thus, maneuvering downward may or may not be successful until the sphincteric ring is reached when all is torn from grasp just as one is about to succeed.

Some kind of a speculum is tried sooner or later. It usually falls short of expectation in facilitating grasping and proves utterly useless for even beginning the downward course. Unavoidable pressure of its upper end against bowel wall is found to work directly counter to any downward pull of the grasping forceps. Thus the speculum itself prevents prolapse of the bowel wall necessary to the delivery of anything attached to that wall. Withdrawal of the speculum after grasping immediately reimposes unaltered the obstacles to descent.

While our unrewarded search of the literature has not been exhaustive it has been more so than would be likely in the instances of most surgeons anticipating such operations. At all events common knowledge of a serviceable technique is wanting, and we therefore present one which has proved simple and effective. By its use one can quickly determine the possibility of delivery and accomplish it expeditiously if achievable.

Fig. 1. Rectal tumor is being grasped through electrically lighted operating rectoscope by ordinary sponge holding forceps. The tips of the latter may be extended beyond the rectoscope for more freedom of manipulation.

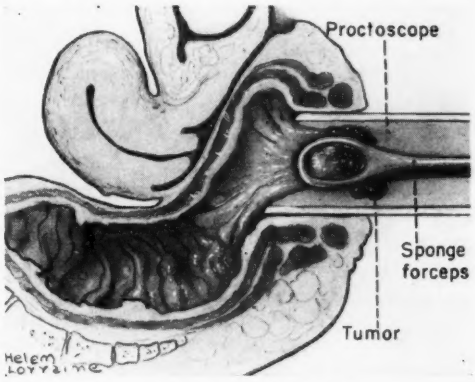

Fig. 2. The tumor has been drawn into the rectoscope as far as the cone shaped base of the bowel wall will permit. Here it lies safely protected inside the rectoscope during passage through the anal canal. Thus the chief interference to delivery is thwarted.

As prelude, analysis of our experience in delivering tumors, such as we have described, indicated that devices aimed at grasping the tumor

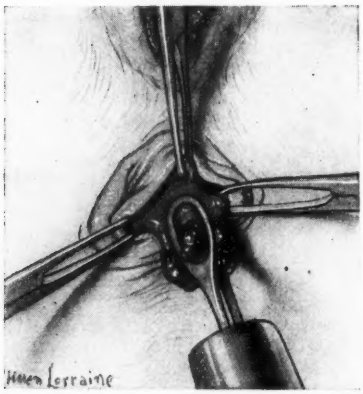

Fig. 3. Allis forceps have firmly grasped bowel wall in each quadrant just above tumor. Releasing sponge forceps will then finally leave lesion in their grasp alone. Tension upward of bowel wall is thus more easily counteracted without tearing by this division into 3 fractions.

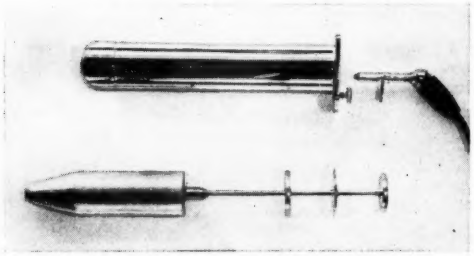

Fig. 4. Operating rectoscope designed for anal delivery of rectal tumors.

and at neutralizing the obstructive constriction of the anal canal during delivery work directly at odds against the necessary forces of downward traction and vice versa. This has been illustrated in reference to employment of a speculum. In seeking a solution one could therefore logically begin with either of the two phases and progress toward the other. As grasping the tumor is certainly the first essential we commenced there.

For facility in grasping a tumor nothing compared in our experience with working through a completely tubal speculum. We therefore designed an operating rectoscope or proctoscope specifically for this purpose. Conditions dictated that it should be of length necessary to grasp a tumor at the highest level offering prospect of delivery. It must be large enough in diameter to admit the necessary grasping instrument yet small enough to pass the sphincter without injury. In these details it is paramount to appreciate that very small variations of diameter in either way can render the instrument useless. One cannot do with *almost* the right instrument.

Our rectoscope is an electrically lighted tube 6 inches long and 1⅛ inches inside diameter, requiring of course anesthesia for introduction. An ordinary straight sponge forceps was found to serve better than anything else tried for firmly seizing the tumor. The blades of this instrument are ⅝ inch in width so that they can be manipulated within the rectoscope.

It is important in carrying out the procedure to include the base of the tumor within the circle of the sponge forceps because the tumor tissue itself may be too friable. If the tips of the blades do not separate widely enough within the rectoscope they can be pushed beyond its end (Fig. 1), permitting wider separation and freer manipulation.

The use of these instruments, then, marked the point of departure for further study of the problem which was simply solved as follows:

Once the tumor base is firmly grasped it is drawn inside the rectoscope. The rectoscope and forceps are then slowly withdrawn *together* as one instrument and with no change whatsoever in relationship to each other. If traction on the two instruments is thus rigidly maintained the tumor will be successfully delivered if it is at all possible. During passage through the anal canal it lies safely protected inside the rectoscope (Fig. 2), and the chief impedence to delivery is thus entirely counteracted. Even when the tumor emerges outside the anus it is still within the rectoscope. Immediately, however, the ends of the forceps are allowed to project by final traction on the rectoscope alone.

The resulting situation is patently a clumsy status, so that 4 Allis forceps are next placed just above the tumor in each quadrant (Fig. 3). While these are held by assistants the sponge forceps, having fulfilled its purpose, is released. The lesion is now finally held by the Allis forceps in excellent position and under full control for inspection and diagnosis and for surgical disposition if so indicated.

SUMMARY

The anal delivery of certain rectal tumors is the most effective method of approach, within its indications, for diagnostic and surgical disposition. A technique has been described which considerably enhances prospects of accomplishment and attendant clinical gains.

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THE SURGEON'S LIBRARY

REVIEWS OF NEW BOOKS

IN HIS book, *Acute Renal Failure*,[1] the author has written a very timely monograph on the much confused subject. He has correlated the work of experts in this field as well as a record of his own personal experiences, and has set down in clear, simple fashion what is known and what is not known concerning the entire problem of acute renal failure.

One of the most important sections of this book is the detailed instructions given on how the patient should be treated, commencing from the moment the diagnosis is made until recovery occurs. Special emphasis is placed on the importance of fluid and electrolyte replacement only to the extent of actual loss, and the importance of watching for and guarding against acidosis. The various methods of removing excess waste products from the blood are discussed with a critical analysis of their usefulness and limitations. The use of the artificial kidney is described in detail, and what one may hope to accomplish by its use. The various types of artificial kidneys available are also described. In this section the author has recorded many personal cases and has shown graphically what can be achieved with this method of treatment.

This book is to be highly recommended to the student, general practitioner, and specialist, to assist him through a maze of conflicting opinions and to assist him in the care of his patients.

HAROLD L. METHOD

THE names of the two authors alone should enlist the keenest interest in the remarkable volume *Die gesunde und die kranke Wirbelsaeule in Roentgenbild und Klinik*.[2] It contains an overwhelming wealth of material as well as a splendid documentation by almost 400 superbly finished illustrations.

It consists of 9 chapters to which is added a bibliography of almost fantastic dimensions. The first chapter deals with the normal growth and structure of the spinal column. A thorough, systematic description acquaints the reader with all phases and steps of spinal development from the newborn to full adult life. This is supported, as is the entire book, by excellent illustrations from roentgenograms and anatomic specimens, side by side. Special attention should be called to the section which deals with the anatomy of the intervertebral disc and to injected specimens which show the normal and diseased nucleus.

Congenital anomalies are the subject of Chapter 2. Beginning with the developmental background, the authors present beautifully illustrated examples of wedge and cleft vertebrae, synostosis and spondylolysis of the posterior column, and lateral and posterior clefts, as well as numerical variations and assimilation vertebrae, with particular emphasis given to sacralization.

There is a real wealth of information on the morbid changes of the spine contained in the large Chapter 3. It starts with the section on osteoporosis and osteodystrophies, the former including the senile and marantic as well as the rhachitic and osteomalacic types. The osteodystrophic section covers Paget's disease and the osteitis fibrosa generalisata of Recklinghausen and Sudeck's atrophy. Then follows a short section on the less common dystrophies, such as marble bone, osteopoikilosis, cretinism, and acromegaly, with a rather perfunctory mention of myositis ossificans progressiva. Achondroplasia, osteopsathyrosis, platyspondylia, and Calvé's vertebra plana are not omitted. The reader will appreciate particularly a short section on osteopathies in blood dyscrasias in which the authors include myeloma. One will find these sections most interesting reading and unusually documented. Of special usefulness are the paragraphs on vertebral fractures from lesser traumas, from muscle spasm, as well as the much disputed Kümmell's disease, and the marginal fractures and separations of the cartilaginous endplates. The same careful attention is given to the injuries of the posterior column and fractures of the arches of the apophyseal articular, spinous and transverse processes.

In the sections on infections, tuberculosis and osteomyelitis occupy a prominent place, as is to be expected, but the less common infections such as syphilitic spondylitis and mycotic infections are by no means overlooked. Benign tumors such as hemangiomas and osteomas as well as malignant tumors, sarcoma, chordoma, and metastatic lesions are well described and equally well illustrated.

The reader will particularly appreciate what is presented concerning the intervertebral articulations, their inflammatory changes and the loss of patency due to arthritis and osteochondrosis. Although all these subjects are of unusual interest, the

[1] ACUTE RENAL FAILURE; INCLUDING THE USE OF THE ARTIFICIAL KIDNEY. By John T. MacLean, M.D., F.R.C.S. (C)., F.A.C.S. Springfield, Ill.: Charles C Thomas, 1952.

[2] DIE GESUNDE UND DIE KRANKE WIRBELSAEULE IN ROENTGENBILD UND KLINIK. PATHOLOGISCH-ANATOMISCHE UNTERSUCHUNGEN. By Geh. Med.-Rat Prof. Dr. Med. and Dr. Med. Vet. H. C. Georg Schmorl. FUER ROENTGENKUNDE UND KLINIK. By Prof. Dr. Med. Habil. Herbert Junghanns. 3rd enl. ed. Stuttgart: Georg Thieme, 1953.

center of attention undoubtedly is occupied by Chapter 4 which deals with the morbid changes of the intervertebral disc and occupies no less than 60 pages. Some of the highlights are as follows: the expanded high disc in osteoporosis, the narrow disc found in degeneration with or without herniation. The intervertebral penetration of the disc in the form of Schmorl's nodes is given special attention and this leads up to the discussion of the notorious posterior prolapse, which includes interestingly enough also the mentioning of herniation following lumbar puncture. In this connection the authors make some very relevant observations regarding differential diagnosis. One of these is the presence of crests at the posterior edge of the vertebral body, another is Luschka's uncovertebral joints of the cervical spine, both of which can produce impingement symptoms, simulating a herniated disc. What the clinician should appreciate most is a short but extremely punctilious discussion of the spontaneous disappearance of root symptoms in connection with the fibrous repair of the degenerated disc, be it prolapsed or not. No less instructive is the section on the degeneration of the disc in relation to osteochondrosis of the spine, and illustrations showing calcification of the disc and osseus formations at the vertebral edges are of excellent quality. Trauma and destructive inflammatory lesions of the vertebral column are treated next and short mention is made of metastatic tumors.

Chapter 5 deals with spondylolysis deformans, mechanically, roentgenologically, and clinically. It is less voluminous and yet, so far as we can see, it is a complete presentation. So also is the following Chapter 6 on the subject of kyphotic and scoliotic deformations of the spine and Chapter 7 which deals with ankylosing spondylitis. Chapter 8 is of more than passing interest. It is devoted to the sliding displacements of the vertebra. True spondylolisthesis has been discussed previously in Chapter 2 with congenital anomalies. In this chapter the authors consider the posttraumatic type which follows symetrical fracture of the isthmus (pars interarticularis) as well as the displacements due to arthritic changes of the articulations, which the authors classify as pseudospondylolisthesis. In addition they pay attention to the controversial subject of retrospondylolisthesis which they recognize as the result of a downward deflection of the articular processes.

The lumbosacral region is mentioned only in a percursory way in the last chapter (9) since anomalies of this region were given ample space in Chapter 2 on the developmental changes.

That this book represents a classic need hardly be pointed out to anyone who gives even a cursory glance. Some of the sections seem to be in urgent need of being translated especially those that deal with the pathology and clinical description of the protruding disc.

The work is a fitting monument to the great George Schmorl who has laid the foundation to the pathology of the spine on the basis of his enormous experience. Since his death in 1932, the equally prominent co-author Herbert Junghanns, has carried on and prepared a second edition of this work which ranks with the great classics in the field of pathology of the spine. ARTHUR STEINDLER.

THIS carefully written and well illustrated volume is the fruitful, scholarly result of many years of study, dissection, and surgical observations. *Functional and Surgical Anatomy of the Hand*[1] deserves careful reading by everyone who is interested in the surgical treatment of injuries and pathologic conditions affecting the hand.

The opening chapter upon the function of the hand—"The Hand as an Organ," is followed by Part 2 with detailed and well illustrated descriptions of the bones, joints, muscles, tendons, nerves, and fascias of the hand. The final chapter of Part 2, No. 7, is devoted to a detailed discussion of the mechanism of action of the fingers, thumb, and wrist, a subject to which the author has devoted much study and which he has helped to clarify and make understandable both to students and surgeons. His detailed description and analysis of the complicated extensor mechanism of the fingers, in particular, is an outstanding addition to our understanding of the function of the motors of the fingers.

In Chapter 2, his description of the chiasma tendinum of Camper calls attention to an important and not too well known anatomic structure of particular importance to the surgeon when he attempts to draw the flexor sublimis from a finger. One may not realize that unless the chiasma is divided longitudinally (or ruptured by brute strength), it is impossible to draw the sublimis proximally from the finger even though both slips of insertion have been completely divided.

In Chapter 6 on the "Retinacular System of the Hand" the author correctly says that the terms "thenar space" and "midpalmar space" are not truly applicable to the locations where pus accumulates on rupture from the tendon sheaths of the fingers. He suggests the term "adductor space" instead of "thenar space," or perhaps "deep retrotendinous radial and retrotendinous ulnar palmar spaces" as definitely descriptive. Possibly the very simple terms "lateral palmar space" and "medial palmar space" might be adequate and satisfactory.

In connection with this subject the author says "There are no septa from the midpalmar to the fascia of the deep volar aspect of the hand," and "No septum between the fascia and the crest of the third metacarpal is in existence." In Figure 42 and Figure 75, however, a definite partition is shown which separates the adductor or thenar space from the midpalmar space. The author states that "the separation is due mostly to adherence of the ulnar bursa to the crest of the third metacarpal." This

[1]FUNCTIONAL AND SURGICAL ANATOMY OF THE HAND. By Emanuel B. Kaplan, M.D., F.A.C.S. Philadelphia, London Montreal: J. B. Lippincott Co. 1953

subject is mentioned only because so many conflicting opinions have been expressed by various writers. One wonders if these differences of opinion do not result from different interpretations of words like "septum", and "partition." One cannot gainsay the fact that the two palmar spaces are definite, and definitely separated one from the other. In an experience of many years on the attending staff of the Cook County Hospital in an era when infections of the hand were exceedingly common and often of prolonged duration this reviewer cannot recall a single instance where an infection of the thenar space had ruptured or extended into the midpalmar space, or vice versa. Such a case is illustrated in Kanavel's "Infections of the Hand," but the writer has never observed one himself.

With reference to Dupuytren's contracture, the author says "The fascia in the hand afflicted with Dupuytren's contracture is not a normal fascia and does not recapitulate or exaggerate a normal condition." One must agree that the fascia is not normal, but can one logically say that the thickening and contraction are not the result of a disease process affecting a normal structure? It is difficult to believe that the individual with Dupuytren's contracture is constituted differently from the rest of his fellows.

Part 3, Chapters 8, 9, and 10, is devoted to surface anatomy of the hand and wrist, surgical approaches, and a brief discussion (8 pages) of amputations and substitution of an index for the thumb. Here again excellent illustrations add clearness and understanding in admirable fashion.

Surgeons in general and men working with injured hands in particular are greatly indebted to Dr. Kaplan for his excellent and helpful contribution. A careful study of this volume will aid them to carry out better and more meticulous operative procedures.

SUMNER L. KOCH.

IN THESE days of preventive inoculation, immunization, antibiotic therapy, and rigid medical inspection, it is difficult to realize and visualize the paralyzing effect of epidemic disease on every phase of life during the early years of our nation. Duffy's interesting and well documented *Epidemics in Colonial America*[1] is a vivid and factual account of the major epidemics of smallpox, diphtheria, scarlet fever, yellow fever, and other infectious diseases during the seventeenth and eighteenth centuries.

The author's account of the introduction and results of the practice of variolation (protective inoculation with pus from smallpox patients) recounts a phase of the history of the disease not widely known. "There is no question that smallpox inoculation was a great boon to eighteenth-century Americans. Although some improvement was made in medical treatment, variolation was the major factor in reducing fatalities from smallpox as the century advanced. Admittedly, uncontrolled inoculation was responsible for spreading the infection in the first 30 or 40 years of its use, but smallpox epidemics steadily lessened in intensity, and with this decline came a corresponding diminishment of the terror which this plague had formerly aroused. . . . Not only did variolation serve as a relatively effective check upon smallpox in the colonies, but it also paved the way for the immediate acceptance of vaccination both in England and in America . . . when Edward Jenner made his revolutionary discovery of vaccination at the close of the century, his method was immediately accepted. The way for vaccination had been well prepared, and during his lifetime Jenner was awarded the honors and distinctions that had come to most of his predecessors only posthumously" (pp. 41, 42).

How far reaching and often disastrous an effect epidemic disease produced in the life of the American colonies, and some of its remote results are indicated by a few direct quotations.

"The main attack (of smallpox) hit Charlestown during the winter of 1677–1678, but in Boston the peak of the epidemic was not reached until the following September. On June 6 (1678), a public fast was decreed, for by this time more than 80 deaths had occurred in the two towns. It proved of little avail: Increase Mather stated in his diary that, by the end of July, 7 or 8 people were dying each week in Boston alone. By September the deaths had surged upwards; 30 persons died in one day, and 150 within a four-week period" (p. 46).

"Smallpox had a direct effect upon education in the colonies both for good and evil. Lack of higher educational facilities compelled the colonists to visit England in order to complete their education . . . many of those who did so fell victims to smallpox or other diseases and never returned. From this high mortality among American travelers abroad came the development of institutions of higher learning, such as the College of William and Mary. . . . The threat of smallpox in England was a decided stimulus to the development of colleges in the colonies" (pp. 108, 109). (The latter statement, however, seems more accurate than the first, for the development of institutions of higher learning has always followed emigration from an older civilization.)

Concerning yellow fever, "Among yellow-fever victims in Charleston were the chief justice, receiver-general, provost marshal, and almost half of the assembly. With half the governing officials ill and the remainder incapacitated from fear of the disorder, the local administration did little to alleviate the general confusion and distress. So many deaths occurred that the dead were simply piled into carts; all business activity ceased for five weeks, and nothing was done 'but carrying Medicines,digging graves, (and) carting the dead'."

"The outbreak in Philadelphia was on a comparable scale with that in Charleston. As in Charleston, community life came almost to a standstill. A Quaker diarist reckoned that 'In this distemper had died six, seven, and sometimes eight in a day, for

[1] EPIDEMICS IN COLONIAL AMERICA. By John Duffy. Baton Rouge: Louisiana State University Press, 1953.

several weeks, there being few houses, if any, free of the sickness. Great was the fear that fell on all flesh! (He) saw no lofty or airy countenances nor heard any vain jesting to move men to laughter. . . . But every face gathered paleness, and many hearts were humbled, and countenances fallen and sunk, as such that waited every moment to be summoned to the bar and numbered to the grave'" (pp. 143, 144).

If one becomes discouraged at times that many medical problems remain unsolved, he can take courage as he considers the advances that have been made since the fearsome days when epidemics of infectious disease were so frequent and disastrous, days so well portrayed in Duffy's historical account of some of the difficulties and tragedies associated with the birth of our nation.

SUMNER L. KOCH.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

ANNALS OF ROENTGENOLOGY. A SERIES OF MONOGRAPHIC ATLASES. Vol. 21. TUMORS: A ROENTGENOGRAPHIC ATLAS. By Bradley L. Coley, M.D., F.A.C.S., and Norman L. Higinbotham, M.D., C.M., F.A.C.S. 216 pages, with 172 illustrations. $10.00. New York: Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, 1953.

CHIRURGIE DU NOUVEAU-NÉ ET DU NOURRISSON. By Bernard Duhamel. Preface by Pr Marcel Fènre. 308 pages, with 158 illustrations. Paris: Masson & Cie, 1953.

ROSE AND CARLESS' MANUAL OF SURGERY FOR STUDENTS AND PRACTITIONERS. By Sir Cecil Wakeley, BT., K.B.E., C.B., LL.D., M.Ch., D.Sc., F.R.C.S., F.R.S.E., F.R.S.A., F.A.C.S., F.R.A.C.S. Assisted by 18 contributors. 18th ed. 1,471 pages, with 1,011 illustrations. $12.00. Baltimore: The Williams & Wilkins Co., 1952.

SHOULDER LESIONS. By H. F. Moseley, M.A., D.M., M.Ch. (Oxon.), F.R.C.S. (England & Canada), F.A.C.S. 2nd rev. enl. ed. 329 pages, with 216 illustrations. $12.00. New York: Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, 1953.

SECTIONAL RADIOGRAPHY OF THE CHEST. By Irving J. Kane, M.D. Foreword by Edward D. Churchill, M.D. 154 pages, with 101 illustrations. $7.50. New York: Spring Publishing Co., Inc., 1953.

GYNAEKOLOGISCHE ORGANNEUROSEN; EINFUEHRUNGSVORLESUNGEN IN DIE PSYCHOSOMATIK. By Prof. Dr. Hans Roemer. 190 pages. DM 13.50. Stuttgart: Georg Thieme, 1953.

ROENTGEN ANATOMY; ROENTGEN ANATOMÍA. By David Steel, M.D. 109 pages, illustrated. $8.00. Springfield, Ill.: Charles C Thomas, 1951.

PROCEEDINGS OF THE SOCIETY FOR THE STUDY OF FERTILITY. No. IV. London Conference, 1952. 63 pages, illustrated. 10 shillings. Cambridge: W. Heffer & Sons Ltd., 1953.

TWENTY-FIVE YEARS OF SEX RESEARCH: HISTORY OF THE NATIONAL RESEARCH COUNCIL COMMITTEE FOR RESEARCH IN PROBLEMS OF SEX, 1922–1947. By Sophie D. Aberle and George W. Corner. 248 pages. $4.00. Philadelphia, London: W. B. Saunders Co., 1953.

SURGERY OF THE PANCREAS. By Richard B. Cattell, M.D., and Kenneth W. Warren, M.D. 374 pages, with 100 illustrations. $10.00. Philadelphia, London: W. B. Saunders Co., 1953.

THE SURGERY OF INFANCY AND CHILDHOOD; ITS PRINCIPLES AND TECHNIQUES. By Robert E. Gross, M.D., D.Sc. 1,000 pages, with 1,488 illustrations. $16.00. Philadelphia, London: W. B. Saunders Co., 1953.

EFFECTIVE INHALATION THERAPY. By Edwin Rayner Levine, M.D. With the co-operation of Alvan L. Barach, M.D., J. Winthrop Peabody, M.D., and Maurice S. Segal, M.D. 159 pages, illustrated. $4.50. Chicago: National Cylinder Gas Co., Medical Division, 1953.

MAN'S BACK. By Theodore A. Willis, M.D., F.A.C.S 161 pages, with 125 illustrations. $9.50. Springfield, Ill.: Charles C Thomas, 1953.

MODERN CONCEPTS IN MEDICINE. By Julius Jensen, Ph.D. (Med.), M.R.C.S. (Eng.), L.R.C.P. (Lond.). 636 pages, illustrated. $11.50. St. Louis: The C. V. Mosby Co., 1953.

ARMED FORCES MEDICAL LIBRARY CATALOG, 1952. (Part 1: Authors; Part 2: Subjects). 813 pages. $17.50. Washington: The Library of Congress, 1953.

ESSENTIAL UROLOGY. By Fletcher H. Colby, M.D. 2nd ed. 650 pages, with 352 illustrations. $8.00. Baltimore: The William & Wilkins Co., 1953.

BLOCKADE AND CHIRURGIE DES SYMPATHICUS. By Dr. Felix Mandl. 388 pages, with 63 illustrations. $12.40. Wien: Springer-Verlag, 1953.

THE SULPHONAMIDES AND ANTIBIOTICS IN MAN AND ANIMALS. By J. Stewart Lawrence, M.D. (Ed.), M.R.C.P., and John Francis, M.Sc., M.R.C.V.S. with the assistance of Arnold Sorsby, M.D., F.R.C.S., and Philip G. Scott, F.R.C.S. 482 pages, illustrated. 42 shillings. London: H. K. Lewis & Co. Ltd., 1953.

URONEFROSIS POR OBSTRUCCION PIELO—URETERAL; TRATAMIENTO PLÁSTICO. By Dr. Ricardo Bernardi. 101 pages, with 66 illustrations. Buenos Aires, 1952.

DIE FUNKTIONELLE HARNINKONTINENZ DER FRAU MIT BESONDERER BERUECKSICHTIGUNG NEUER DIAGNOSTISCHER METHODEN. By Priv.-Doz. Dr. Hubert Hartl. 99 pages, with 36 illustrations. DM 10. Stuttgart: Ferdinand Enke Verlag, 1953.

THE BOOK OF HEALTH; A MEDICAL ENCYCLOPEDIA FOR EVERYONE. Edited by Randolph Lee Clark, Jr., B.S., M.D., M.Sc. (Surg.), and Russell W. Cumley, B.A., M.A., Ph.D. 1st ed. 768 pages, with 1,400 illustrations. $10.00. Houston, New York: Elsevier Press Inc., 1953.

MECHANISMS OF UROLOGIC DISEASE. By David M. Davis, M.D. 156 pages, illustrated. $4.50. Philadelphia, London: W. B. Saunders Co., 1953.

HUMAN NEUROANATOMY. By Oliver S. Strong and Adolph Elwyn. 3rd ed. 481 pages, with 357 illustrations. $7.50. Baltimore: The Williams & Wilkins Co., 1953.

A HANDBOOK OF RADIOTHERAPY. By Walter M. Levitt, M.D., F.R.C.P. (Lond.), F.F.R., D.M.R.E. (Camb.). 232 pages, with 53 illustrations. $6.50. New York: Paul B. Hoeber, Inc., Medical Book Dept. of Harper & Bros., 1953.

THE PRACTICE OF BALANCED ANESTHESIA. By Sylvan M. Shane, D.D.S. 196 pages, with 12 illustrations. $5.75. Baltimore: Lowry & Volz, 1953.

CLINICAL CONGRESS OF AMERICAN COLLEGE OF SURGEONS

HAROLD L. FOSS, Danville, Pennsylvania, *President*
FRED W. RANKIN, Lexington, Kentucky, *President-Elect*

PRELIMINARY PLANS FOR 39TH CLINICAL CONGRESS, THE CONRAD HILTON, CHICAGO, OCTOBER 5 TO 9, 1953

THE SIGNIFICANCE of the Clinical Congress of the American College of Surgeons to the progress of surgery in America will be demonstrated again in Chicago, October 5 to 9. Of far-reaching importance in influencing the future of surgery, the Congress utilizes many methods and is constantly devising new ones to further the education of the surgeon. In its years of hard, earnest work to further College aims, under aggressive leadership and uncompromising integrity, the aim of the Congress to increase efficiency and raise the standard of every surgeon shows evidence of wide approval: 11,526 came to the meeting in New York in 1952, and it is anticipated that attendance will again reach an equally high number in 1953.

The general plan of the Congress is the same as that of previous years. Deviations from earlier programs have been made with the interests of the surgeons in mind: because of the superiority of the motion picture, ciné clinic, and television processes in permitting more surgeons to view operative techniques, wet clinics have been eliminated this year. Also, because the American Boards of Ophthalmology and Otolaryngology are holding examinations during Congress week, the College's Advisory Councils for Ophthalmology and Otolaryngology have recommended that sessions in these two specialties be limited to three evening programs, at which important panel discussions have been planned.

POSTGRADUATE COURSES

Six postgraduate courses will be given mornings, from 8:30 until 11:30, on Tuesday through Friday. Because seating space for these popular courses will be limited it is important to make reservations in advance. Registration forms may be obtained now from the American College of Surgeons, 40 East Erie Street, Chicago 11, Illinois. The fee is $5.00 per course. The program appears on following pages.

COLOR TELEVISION

Color television programs, made possible by Smith, Kline & French Laboratories of Philadelphia, will emanate from Albert Merritt Billings Hospital and will be shown at the Conrad Hilton. Operation schedules will be published in the *Daily Bulletin* of the Congress. The program, under the leadership of Dr. Lester R. Dragstedt, will consist of surgical procedures and related clinical material carefully integrated with the entire Congress program. New equipment, added this year, promises a better picture than ever before.

CINÉ CLINICS

Ciné clinics will be held Tuesday through Friday mornings. Late developments in the field of surgery and standard procedures in the various surgical specialties will be covered in the sessions. Under the guidance of Dr. Hilger P. Jenkins, Chairman, and his Medical Motion Picture Committee, the following program has been planned:

Creation of an Artificial Ductus in the Treatment of the Tetralogy of Fallot. ALFRED BLALOCK, M.D., F.A.C.S., Chicago.

Reconstruction of Common Bile Duct. I. S. RAVDIN, M.D., F.A.C.S., Philadelphia.

Hernioplasty. LEO M. ZIMMERMAN, M.D., F.A.C.S., Chicago.

Vagotomy and Gastrectomy. LESTER R. DRAGSTEDT, M.D., F.A.C.S., Chicago.

Pneumonectomy. WILLIAM E. ADAMS, M.D., F.A.C.S., Chicago.

Pancreatic Cysts. CHARLES B. PUESTOW, M.D., F.A.C.S., Chicago.

Ovarian Cysts. HERBERT E. SCHMITZ, M.D., F.A.C.S., Chicago.
Tendon Injuries. MICHAEL L. MASON, M.D., F.A.C.S., Chicago.
Surgery of the Aged. ERWIN R. SCHMIDT, M.D., F.A.C.S., Madison.
Endometriosis. EDWARD D. ALLEN, M.D., F.A.C.S., Chicago.
Diverticulitis. R. KENNEDY GILCHRIST, M.D., F.A.C.S., Chicago.
Thyroidectomy. FREDERICK A. COLLER, M.D., F.A.C.S., Ann Arbor.
Intestinal Obstruction. CHARLES G. JOHNSTON, M.D., F.A.C.S., Detroit, and RUDOLF J. NOER, M.D., F.A.C.S., Louisville.
Diabetic Foot. ANGUS D. MACLACHLIN, M.D., F.A.C.S., London, Ontario.

MEDICAL MOTION PICTURE PROGRAM

Congress visitors are unusually fortunate this year. An outstanding program of approximately thirty new films, showing rare surgical conditions, with the operating surgeons present at the sessions, has been planned by the Committee on Medical Motion Pictures. Special attention is called to the showing of an important teaching film just made at Harvard University, "The Repair of Inguinal Hernia," the first in a series of films to be entitled "The Surgical Residency Series of Teaching Motion Pictures." This series, made under the direction of Dr. Francis D. Moore, will explore the technical problem in some detail.

Films on fractures, from a new series being produced by the Veterans Administration, will be an added feature of the program. In addition special otolaryngology and ophthalmology films will be shown at their individual sessions.

On Thursday evening at 8 p.m. the Motion Picture Symposium on "Unusual Conditions Encountered in Abdominal Surgery" will be held. Details of this important symposium will be announced in the *Daily Bulletin* of the Congress.

FORUM ON FUNDAMENTAL SURGICAL PROBLEMS

Twelve Forum sessions have been planned for this year. Details are set forth in the preliminary printed program in this issue. Chairman of the Surgical Forum Committee is Dr. I. S. Ravdin.

AFTERNOON AND EVENING SESSIONS

Three general evening sessions have been planned, on Monday, Wednesday, and Friday. Tuesday and Thursday evenings are open for special meetings. The meeting on Monday evening will feature an address by Dr. Harold L. Foss, retiring President, and the Eighth Martin Memorial Lecture by Sir James Paterson Ross, London, England, speaking on "Science and Surgery." Dr. Robert H. Kennedy will preside over the Wednesday evening scientific session, which will include reports by Dr. J. William Hinton, New York, Dr. Francis D. Moore, Boston, and the Oration on Trauma by Dr. William L. Estes, Jr., Bethlehem, Pennsylvania. On Friday evening new Fellows will be received into the College, and Honorary Fellowships conferred. New President Dr. Fred W. Rankin will make his incoming Presidential Address.

ADVANCE RESERVATIONS

Scientific sessions will begin promptly on Monday morning; hence it is advisable to complete the registration process in advance. Official registration forms are available from the American College of Surgeons. This advance registration involves only a brief check-in on arrival. Registration desk hours have been increased as follows: Sunday, October 4, from 3 p.m. to 9 p.m.; Monday, October 5, from 8 a.m. to 7:30 p.m.; Tuesday through Friday, 8 a.m. to 5 p.m.

No registration fee will be charged Fellows whose dues are paid to December 31, 1952; no registration fee is required of Initiates, Junior Candidates or Surgical Residents. Non-Fellows and Fellows whose dues are not paid will pay a fee of $20.00. It is also suggested that non-Fellows bring medical identification with them, to simplify their registration.

HOTEL ACCOMMODATIONS

For the most desirable hotel accommodations it is advisable to make advance reservations. Official forms or information may be obtained from the American College of Surgeons.

PROGRAM IN BRIEF

Monday, October 5, 1953

9:00–12:00 Forum: Lungs and Peripheral Circulation
9:00–12:00 Medical Motion Pictures
9:00–12:00 Forum: Heart
9:00–12:00 Television
10:30–12:00 Panel Discussion
1:30– 3:00 Clinicopathology Conference
1:30– 5:30 Medical Motion Pictures
2:00– 4:30 Television
2:00– 5:00 Forum: Burns, Wounds, Anesthesia
2:00– 5:00 Forum: Gynecology and Obstetrics
3:30– 5:00 Panel Discussion
6:30– 7:00 Reception preceding Presidential Dinner
7:00– 8:15 Presidential Dinner—Board of Regents, Wives, Guests
8:30–10:00 Presidential Meeting

Tuesday, October 6, 1953

8:30–11:30 Postgraduate Courses:
Preoperative and Postoperative Care
Surgery of the Upper Gastrointestinal Tract
Surgery of the Small and Large Bowel
Cardiovascular Surgery
Obstetrics and Gynecology
Trauma to the Abdomen
9:00–12:00 Television
9:00–12:00 Forum: Heart and Great Vessels
9:00–12:00 Forum: Pancreas, Liver and Intestines
9:00–12:00 Medical Motion Pictures
9:00–12:30 Ciné Clinic
10:00–12:00 Directors Meeting—S.G. & O.
1:30– 5:30 Medical Motion Pictures
1:30– 3:00 Panel Discussion
2:00– 4:30 Television
2:00– 5:00 Symposium on Trauma
2:00– 5:00 Forum: Cancer and Endocrine Glands
2:00– 5:00 Forum: Plastic Surgery
3:00– 5:00 Library Committee
3:30– 5:00 Panel Discussion
5:00– 6:00 International Society of Surgery
6:30– 8:00 Dinner for Speakers on Evening Programs
6:30–10:00 Dinner—3rd Auxiliary Surgical Group
6:30–10:00 Dinner, Officers of Local Chapters
7:00– 8:00 Medical Motion Pictures (EENT)
8:30–10:30 Scientific Session, Otolaryngology
8:30–10:30 Scientific Session, Ophthalmology

Wednesday, October 7, 1953

8:30–10:30 Breakfast, Committee on Nutrition
8:30–11:30 Postgraduate Courses:
Preoperative and Postoperative Care
Surgery of the Upper Gastrointestinal Tract
Surgery of the Small and Large Bowel
Cardiovascular Surgery
Obstetrics and Gynecology
Trauma to the Abdomen
9:00–12:00 Television
9:00–12:00 Forum: Shock and Plasma Expanders
9:00–12:00 Forum: Gastrointestinal Tract
9:00–12:00 Forum: Urology
9:00–12:30 Ciné Clinic
10:00–12:00 Meeting, Credentials, Judiciary and Counseling Committees
1:30– 3:00 Clinico-Pathology Conference
1:30– 3:00 Panel Discussion: Gynecology and Obstetrics
1:30– 5:30 Medical Motion Pictures
1:30– 5:30 Adjourned Meeting of Governors
2:00– 4:30 Television
2:00– 5:00 Symposium on Cancer
2:00– 5:00 Forum: Orthopedic Surgery
2:00– 5:00 Forum: Nutrition and Electrolytes
3:30– 5:00 Panel Discussion: General Surgery
3:30– 5:00 Panel Discussion: Gynecology and Obstetrics
3:30– 5:00 Meeting: Regional and National Trauma Committees
6:30– 8:00 Dinner for Speakers on Evening Sessions
6:30–10:00 Dinner—4th Surgical Auxiliary Group
7:00– 8:00 Medical Motion Pictures (EENT)
8:30–10:30 Scientific Session
8:30–10:30 Scientific Session: Ophthalmology
8:30–10:30 Scientific Session: Otolaryngology

Thursday, October 8, 1953

7:30– 9:30 Breakfast, Forum Committee
8:30–10:30 Nominating Committee
8:30–11:30 Postgraduate Courses
9:00–12:00 Television
9:00–12:00 Forum: Peripheral Circulation and Shock
9:00–12:00 Forum: Lower Nephron and Blood Coagulation
9:00–12:00 Forum: Neurologic Surgery
9:00–12:30 Ciné Clinic
1:30– 3:00 Panel Discussion: General Surgery
1:30– 3:00 Panel Discussion: Gynecology and Obstetrics
1:30– 3:00 Symposium: Graduate Training in Surgery
1:30– 5:30 Medical Motion Pictures
2:00– 4:30 Television
3:30– 3:45 Adjourned Meeting, Board of Governors
3:30– 6:00 Annual Meeting of Fellows
6:30– 8:00 Dinner, Speakers on Evening Programs
6:30–10:00 Dinner Meeting, Trauma Committee
7:00– 8:00 Medical Motion Pictures (EENT)
8:00–10:00 Medical Motion Picture Symposium
8:30–10:30 Scientific Session: Ophthalmology
8:30–10:30 Scientific Session: Otolaryngology

Friday, October 9, 1953

8:30–11:30 Postgraduate Courses
9:00–12:00 Television
9:00–12:00 Meeting, Board of Regents
9:00–12:00 Medical Motion Pictures
9:00–12:00 Ciné Clinic
12:00– 1:30 Luncheon, Board of Regents
1:30– 3:00 Assembly of Initiates
1:30– 4:45 Panel Discussion:
Gynecology and Obstetrics
Neurologic Surgery
Orthopedic Surgery
Plastic Surgery
Thoracic Surgery
Urology
2:00– 4:30 Television
6:30– 7:00 Reception preceding Regents Dinner
7:00– 8:15 Regents Dinner, Board of Regents, Wives, and Guests
6:30– 7:00 Reception preceding Governors Dinner
7:00– 8:15 Governors Dinner
7:30– 8:15 Initiates Pre-Convocation Assembly
8:15– 8:30 Assembly for Officers, Regents, Governors and Guests
8:30–10:00 Convocation

PRELIMINARY PROGRAM

EVENING SESSIONS

PRESIDENTIAL MEETING

Monday, 8:30–10:00 p.m., Grand Ballroom, Conrad Hilton

HAROLD L. FOSS, M.D., F.A.C.S., Danville, Pennsylvania; President, American College of Surgeons, *Presiding.*

Introduction of Guests. EVARTS A. GRAHAM, M.D., F.A.C.S., St. Louis; Chairman, Board of Regents.

Presidential Address: The American College of Surgeons, Its Obligations and Responsibilities, Never Greater Than Now. HAROLD L. FOSS, M.D., F.A.C.S. (Retiring President).

The Eighth Martin Memorial Lecture: Science and Surgery. SIR JAMES PATERSON ROSS, London; Vice President, Royal College of Surgeons of England.

Recession.

EVENING SCIENTIFIC SESSION

Wednesday, 8:30–10:00 p.m., Grand Ballroom, Conrad Hilton

ROBERT H. KENNEDY, M.D., F.A.C.S., New York, *Presiding.*

Cardiac Arrest; Etiology and Management from a Study of Over 1300 Cases. J. WILLIAM HINTON, M.D., F.A.C.S., New York.

Metabolic and Electrolyte Abnormalities in Severly Ill Cardiac Patients and Individuals with Advanced Cancer. FRANCIS D. MOORE, M.D., F.A.C.S., Boston.

Oration on Trauma: WILLIAM L. ESTES, JR., M.D., F.A.C.S., Bethlehem, Pennsylvania.

CONVOCATION

Friday, 8:30–10:00 p.m., Grand Ballroom, Conrad Hilton

HAROLD L. FOSS, M.D., F.A.C.S., Danville, Pennsylvania; President, American College of Surgeons, *Presiding.*

Procession. Initiates, Governors, Officers, Regents, and Distinguished Guests.

Invocation. Reverend Kenneth Hildebrand, Pastor, The Central Church of Chicago.

Induction of Officers:

Presented by ROBERT H. KENNEDY, M.D., F.A.C.S., New York, Retiring First Vice President.

Second Vice-President: DEAN M. LIERLE, M.D., F.A.C.S., Iowa City.

First Vice-President: FRANK GLENN, M.D., F.A.C.S., New York.

President: FRED W. RANKIN, M.D., F.A.C.S., Lexington, Ky.

Presentation of Initiates for Fellowship. Chairman of the Board of Regents.

Fellowship Pledge.

Conferring of Fellowships by the President.

Conferring of Honorary Fellowships.

Presidential Address: The Responsibility of a Heritage of Idealism. FRED W. RANKIN, (Incoming President).

Recession.

CLINICOPATHOLOGY CONFERENCES

GENERAL SURGERY

Monday, 1:30–3:00 p.m., Grand Ballroom, Conrad Hilton

Moderator: PAUL R. CANNON, M.D., Chicago.

Collaborators: (For first case) JEROME R. HEAD, M.D., F.A.C.S., Chicago; LEROY H. SLOAN, M.D., F.A.C.P., Chicago; FAY HUFFMAN SQUIRE, M.D., Chicago. (For second case) ROBERT ELMAN, M.D., F.A.C.S., St. Louis; FRANK GLENN, M.D., F.A.C.S., New York; JEROME R. HEAD, M.D., F.A.C.S., Chicago; WALTER G. MADDOCK, M.D., F.A.C.S., Chicago; FRANCIS D. MOORE, M.D., F.A.C.S., Boston; FAY HUFFMAN SQUIRE, M.D., Chicago.

Wednesday, 1:30–3:00 p.m., Grand Ballroom, Conrad Hilton

Moderator: WILLIAM B. WARTMAN, M.D., Chicago.

Collaborators: (For both cases) PAUL C. HODGES, M.D., Chicago; WILLIAM D. HOLDEN, M.D., F.A.C.S., Cleveland; JOHN H. MULHOLLAND, M.D., F.A.C.S, New York.

POSTGRADUATE COURSES

PREOPERATIVE AND POSTOPERATIVE CARE

WALTER G. MADDOCK, M.D., F.A.C.S., *Chairman*

Tuesday, 8:30—11:30 a.m., Crystal Ballroom, Blackstone Hotel

The Effect of Operation on Kidney Function. CARL A. MOYER, M.D., F.A.C.S., St. Louis.
Common Mistakes in the Administration of Water and Electrolytes. ROBERT ELMAN, M.D., F.A.C.S., St. Louis.
When Is Oxygen Beneficial? PHILIP B. PRICE, M.D., F.A.C.S., Salt Lake City.
Thromboembolism: a Continuing Problem. MICHAEL E. DE BAKEY, M.D., F.A.C.S., Houston.

Wednesday, 8:30—11:30 a.m., Crystal Ballroom, Blackstone Hotel

Clinical Lessons From Blood Volume Studies. CHAMP LYONS, M.D., F.A.C.S., Birmingham.
Blood, Plasma, Plasma Fractionization Products; Their Uses and Abuses. J. GARROTT ALLEN, M.D., F.A.C.S., Chicago.
Postoperative Pulmonary Complications; Their Avoidance and Treatment. ROBERT M. JANES, M.D., F.A.C.S., Toronto.
Cardiac Arrest and Resuscitation. E. H. FELL, M.D., F.A.C.S., Chicago.

Thursday, 8:30—11:30 a.m., Crystal Ballroom, Blackstone Hotel

Meeting the Supranormal Dietary Requirements of Surgical Patients. JONATHAN E. RHOADS, M.D., F.A.C.S., Philadelphia.
The Effect of Nutritional Management in Recovery from Burns. EVERETT I. EVANS, M.D., F.A.C.S., Richmond.
Problems Peculiar to Surgery of Infants and Children. CLIFFORD D. BENSON, M.D., F.A.C.S., Detroit.
Surgery in the Elderly. CHARLES C. LUND, M.D., F.A.C.S., Boston.

Friday, 8:30—11:30 a.m., Crystal Ballroom, Blackstone Hotel

Traumatic Endocrinology and Hormone Administration. FRANCIS D. MOORE, M.D., F.A.C.S., Boston.
How Gastrointestinal Distention Develops and Its Prevention. WALTER G. MADDOCK, M.D., F.A.C.S., Chicago.
Observations of the Reduction of Intestinal Distention with Long Tubes. RUDOLF J. NOER, M.D., F.A.C.S., Louisville.
The Best Results from Antibiotic Therapy. WILLIAM A. ALTEMEIER, M.D., F.A.C.S., Cincinnati.

SURGERY OF THE UPPER GASTROINTESTINAL TRACT

JOHN T. REYNOLDS, M.D., F.A.C.S., *Chairman*

Tuesday, 8:30—11:30 a.m., Eleventh Street Theatre

Esophagus

Benign Lesions:

Bleeding Esophageal Varicosities. ORMAND C. JULIAN, M.D., F.A.C.S., Chicago.
Tracheo-esophageal Fistulas. N. LOGAN LEVEN, M.D., F.A.C.S., St. Paul.
Esophageal Diverticulum, Pharyngeal and Epiphrenic. GUSTAF E. LINDSKOG, M.D., F.A.C.S., New Haven.
Hiatus Hernia. ANTHONY R. CURRERI, M.D., F.A.C.S., Madison.
Acute Burns of the Esophagus; Benign Strictures of the Esophagus. PAUL H. HOLINGER, M.D., F.A.C.S., Chicago.

Carcinoma of the Esophagus:

Carcinoma of the Upper Third of the Esophagus. DANELY P. SLAUGHTER, M.D., F.A.C.S., Chicago.
Causes of Mortality Following Resection of the Middle and Upper Third of the Esophagus. EDWARD F. PARKER, M.D., F.A.C.S., Charleston.
Palliative Operations for Carcinoma of the Esophagus. MARK M. RAVITCH, M.D., F.A.C.S., Baltimore.
Betatron Therapy for Carcinoma of the Esophagus. ROGER A. HARVEY, M.D., Chicago.

Wednesday, 8:30—11:30 a.m., Eleventh Street Theatre

The Stomach and Duodenum

The Physiology of Gastric Secretion; Its Role in Peptic Ulceration. LESTER R. DRAGSTEDT, M.D., F.A.C.S., Chicago.

Symposium on Duodenal Ulcer: The Rationale of Surgical Approaches to Peptic Ulceration. REGINALD H. SMITHWICK, M.D., F.A.C.S., Boston.

Operations:

1. Vagectomy for Peptic Ulceration. KEITH S. GRIMSON, M.D., F.A.C.S., Durham.
2. Gastrectomy for Duodenal Ulcer. HENRY H. HARKINS, M.D., F.A.C.S., Seattle.

Management of Acute Duodenal Ulcers Near the Region of the Common Duct. HAROLD A. ZINTEL, M.D., F.A.C.S., Philadelphia.

The Problem of Massive Hemorrhage from Peptic Ulcer. ROBERT M. ZOLLINGER, M.D., F.A.C.S., Columbus.

Duodenal Diverticulum. MAX M. ZINNINGER, M.D., F.A.C.S., Cincinnati.

Thursday, 8:30—11:30 a.m., Eleventh Street Theatre

Gastric Problems

Peptic Ulceration of the Cardia and Lower Esophagus. RICHARD H. SWEET, M.D., F.A.C.S., Boston.

Gastric Ulcer; Difficulty of Differentiating from Carcinoma. T. J. WACHOWSKI, M.D., Chicago; RALPH E. DOLKART, M.D., Chicago; H. GLENN BELL, M.D., F.A.C.S., San Francisco.

The Role of Total Gastrectomy for Carcinoma of the Stomach. WILLIAM P. LONGMIRE, JR., M.D., F.A.C.S., Los Angeles.

Pouch Substitutes for the Stomach Following Total Gastrectomy. ARTHUR J. HUNNICUTT, M.D., F.A.C.S., Oakland.

The Cause and Prevention of Unsatisfactory Results Following Gastrectomy: Dumping Syndrome; Marginal Ulcer; Malnutrition. JOHN VAN PROHASKA, M.D., F.A.C.S., Chicago; JOSEPH B. KIRSNER, M.D., F.A.C.P., Chicago.

Friday, 8:30—11:30 a.m., Eleventh Street Theatre

The Biliary Tract and Pancreas

The Anatomy of the Bile Ducts, Duodenum and Pancreas. BARRY J. ANSON, Ph.D. (Med. Sc.), Chicago.

Importance and Significance of Hepatic Function Tests in Jaundiced Patients. RICHARD B. CAPPS, M.D., Chicago.

Indications and Contraindications to Operations on the Biliary Tract. NATHAN A. WOMACK, M.D., F.A.C.S., Chapel Hill.

Surgical Errors in Biliary Tract Surgery. GEORGE CRILE, JR., M.D., F.A.C.S., Cleveland.

Persistent Symptoms Following Cholecystectomy. JOHN H. MULHOLLAND, M.D., F.A.C.S., New York.

Chronic Pancreatitis with Especial Emphasis upon Surgical Relief. I. MIMS GAGE, M.D., F.A.C.S., New Orleans.

The Present Status of Pancreaticoduodenectomy in the Treatment of Carcinoma of the Ampulla, the Head of the Pancreas, and the Bile Ducts. CHARLES GARDNER CHILD III, M.D., F.A.C.S., Boston.

SURGERY OF THE SMALL AND THE LARGE BOWEL

R. KENNEDY GILCHRIST, M.D., F.A.C.S., *Chairman*

Tuesday, 8:30—11:30 a.m., Upper Tower Room, Conrad Hilton

Physiology of Large and Small Bowel. HEINRICH NECHELES, M.D., Chicago.

Congenital Anomalies:

Small Bowel. ROBERT E. GROSS, M.D., F.A.C.S., Boston.

Colon, Rectum and Anus. WILLIS J. POTTS, M.D., F.A.C.S., Chicago.

Hirschprung's Disease. ORVAR SWENSON, M.D., F.A.C.S., Boston.

Intestinal Obstruction:

Diagnosis, Physiology, Fluids, Electrolytes. OWEN H. WANGENSTEEN, M.D., F.A.C.S., Minneapolis.

Tubes and Their Use. CHARLES G. JOHNSTON, M.D., F.A.C.S., Detroit.

Intussusception; Surgical Treatment. EDWIN M. MILLER, M.D., F.A.C.S., Chicago.

Wednesday, 8:30—11:30 a.m., Upper Tower Room, Conrad Hilton

Inflammatory Disease of Small Bowel. RALPH COLP, M.D., F.A.C.S., New York.

Ulcerative Colitis:

Diagnosis and Medical Treatment. WALTER L. PALMER, M.D., Chicago.

Surgical Treatment; Indications for Ileostomy and Total Colectomy. G. GAVIN MILLER, M.D., F.A.C.S., Montreal.

Status of Vagotomy; Ileostomy Care; Various Types of Bags. CLARENCE DENNIS, M.D., F.A.C.S., Brooklyn.

Pregnancy and Ulcerative Colitis. FRED O. PRIEST, M.D., Chicago.
Tuberculosis of Colon, Ileocolitis, Actinomycosis, Amoebic Colitis. RUPERT B. TURNBULL, M.D., F.A.C.S., Cleveland.

Thursday, 8:30—11:30 a.m., Upper Tower Room, Conrad Hilton

Diverticulitis of Colon. CLAUDE E. WELCH, M.D., F.A.C.S., Boston.
Carcinoma of the Colon:
Pathology. R. KENNEDY GILCHRIST, M.D., F.A.C.S., Chicago.
Preoperative Treatment and Treatment of Obstructed Lesions; Carcinoma of Colon to Mid-sigmoid. ROBERT A. SCARBOROUGH, M.D., F.A.C.S., San Francisco.
Low-Lying Lesions and Surgical Treatment of Adherent Lesions. CLAUDE F. DIXON, M.D., F.A.C.S., Rochester, Minn.
Second Look Operation. DAVID STATE, M.D., F.A.C.S., Minneapolis.
Palliative Resection and Care of Those with Inoperable Lesions or with Recurrences and Surgery in the Aged and Poor Risk Patient. EUGENE M. BRICKER, M.D., F.A.C.S., St. Louis.

Friday, 8:30—11:30 a.m., Upper Tower Room, Conrad Hilton

Diagnosis of Anorectal Pathology; Traumatic Injuries; Sphincter Incontinence. ROBERT A. SCARBOROUGH, M.D., F.A.C.S., San Francisco.
Hemorrhoids, Surgical and Injection Treatment; Fissures, Crypts, Ulcers. JERROLD P. NESSELROD, M.D., F.A.C.S., Evanston.
Fistulas, Tuberculosis and Nonspecific; Pruritus Ani, Condyloma Accuminata and Venereal Warts. ROBERT TURELL, M.D., F.A.C.S., New York.
Prolapse, Second and Third Degree. WILLIAM A. ALTEMEIER, M.D., F.A.C.S., Cincinnati.
Anesthesia. MAX S. SADOVE, M.D., Chicago.
Carcimoids, Lymphomas, and Allied Conditions. GEORGE HAAS, M.D., Chicago.

GYNECOLOGY AND OBSTETRICS

HERBERT E. SCHMITZ, M.D., F.A.C.S., *Chairman*

Tuesday, 8:30—11:30 a.m., Mercy Hospital, 2537 South Prairie Avenue

Prolapse of the Uterus and Relaxed Vaginal Outlet
Moderator: JOSEPH L. BAER, M.D., F.A.C.S., Chicago.
Applied Anatomy in Relaxation of the Vaginal Outlet. JOHN W. HUFFMAN, M.D., F.A.C.S., Chicago.
The Nonoperative Treatment of Stress Incontinence in Women. LAWRENCE R. WHARTON, M.D., Baltimore.
Manchester-Fothergill Operation. RALPH A. REIS, M.D., F.A.C.S., Chicago.
Vaginal Hysterectomy for Uterine Prolapse. EDWARD D. ALLEN, M.D., F.A.C.S., Chicago.
Treatment of Enterocele Complicating Prolapse. RALPH E. CAMPBELL, M.D., F.A.C.S., Madison.
Treatment of Vaginal Prolapse. EUGENE A. EDWARDS, M.D., F.A.C.S., Chicago.
Discussion: To be opened by Dr. JOSEPH L. BAER.

Wednesday, 8:30—11:30 a.m., Mercy Hospital, 2537 South Prairie Avenue

Abortion
Moderator: NICHOLSON J. EASTMAN, M.D., Baltimore.
Definition and Classifications of Abortions. JAMES E. FITZGERALD, M.D., Chicago.
Injuries of the Cervix and Lower Uterine Segment Influencing Abortion. LEROY A. CALKINS, M.D., Kansas City.
The Endometrium in Abortion. EDWARD C. HUGHES, M.D., F.A.C.S., Syracuse.
Endocrine Therapy in Abortion. RONALD R. GREENE, M.D., Chicago.
Management of Incomplete Abortion. JOSEPH A. HARDY, M.D., St. Louis.
Septic Abortion. CONRAD G. COLLINS, M.D., F.A.C.S., New Orleans.
Discussion: To be opened by Dr. NICHOLSON J. EASTMAN and Dr. M. EDWARD DAVIS, F.A.C.S., Chicago.

Thursday, 8:30—11:30 a.m., Mercy Hospital, 2537 South Prairie Avenue

What Constitutes Conservative Pelvic Surgery?
Moderator: HOWARD C. TAYLOR, JR., M.D., F.A.C.S., New York.
For Menopausal Bleeding. JOHN I. BREWER, M.D., F.A.C.S., Chicago.
For Cervical Lacerations, Erosions, and Polyps. CLYDE L. RANDALL, M.D., Buffalo.
For Myomata. ERLE HENRIKSON, M.D., F.A.C.S., Los Angeles.
For Infertility Due to Tubal Occlusion. JOHN ROCK, M.D., F.A.C.S., Brookline.
For Ovarian Tumors. GEORGE H. GARDNER, M.D., F.A.C.S., Chicago.

For Endometriosis. JOE V. MEIGS, M.D., F.A.C.S., Boston.
For Pelvic Inflammatory Disease. ROBERT A. ROSS, M.D., F.A.C.S., Chapel Hill.
Discussion: To be opened by Dr. HOWARD C. TAYLOR, JR.

Friday, 8:30—11:30 a.m., Mercy Hospital, 2537 South Prairie Avenue

Surgical Complications of Pregnancy

Moderator: SAMUEL A. COSGROVE, M.D., F.A.C.S., Jersey City.
Acute Abdomen. FRANKLIN L. PAYNE, M.D., F.A.C.S., Philadelphia.
Ruptured Uterus. JOHN H. MOORE, M.D., F.A.C.S., Grand Forks.
Lacerations of Cervix, Vagina, and Vulva. JANET E. TOWNE, M.D., F.A.C.S., Chicago.
Carcinoma of the Breast. PETER A. NELSON, M.D., F.A.C.S., Chicago.
Benign Breast Lesions. ROBERT L. SCHMITZ, M.D., F.A.C.S., Chicago.
Indications for Cesarean Hysterectomy. HAROLD L. GAINEY, M.D., F.A.C.S., Kansas City.
Ectopic Gestation. C. GORDON JOHNSON, M.D., F.A.C.S., New Orleans.
Adrenal New Growths. ARTHUR B. HUNT, M.D., Rochester, Minnesota.
Bleeding in the Third Trimester. DAVID N. DANFORTH, M.D., F.A.C.S., Evanston.
Discussion: To be opened by Dr. SAMUEL A. COSGROVE.

CARDIOVASCULAR SURGERY

EGBERT H. FELL, M.D., F.A.C.S., *Chairman*

Tuesday, 8:30—11:30 a.m., Kimball Hall, 306 South Wabash Avenue

Surgical Problems Concerning the Peripheral Arterial System

Moderator: GEZA DE TAKATS, M.D., F.A.C.S., Chicago.
Care of the Arteriosclerotic Limb. NELSON W. BARKER, M.D., Rochester, Minn.
Treatment of Complications of the Arteriosclerotic Limb. LELAND S. MCKITTRICK, M.D., F.A.C.S., Bookline.
Acute Peripheral Arterial Occlusion. NORMAN E. FREEMAN, M.D., F.A.C.S., San Francisco.

Surgical Problems Concerning the Peripheral Venous System

Moderator: J. ROSS VEAL, M.D., F.A.C.S., Washington, D. C.
The Treatment of Thrombophlebitis and Its Complications. WILLIAM D. HOLDEN, M.D., F.A.C.S., Cleveland.
The Treatment of Varicose Veins. THOMAS T. MYERS, M.D., Rochester, Minn.
The Use of Anticoagulants in Vein Pathology. JOHN H. OLWIN, M.D., F.A.C.S., Chicago.

Wednesday, 8:30—11:30 a.m., Kimball Hall, 306 South Wabash Avenue

Aneurysms and Arteriovenous Fistulas

Moderator: HARRIS B. SHUMACKER, JR., M.D., F.A.C.S., Indianapolis.
Participants: FRANK L. GERBODE, M.D., F.A.C.S., San Francisco; WILLIAM S. DYE, M.D., Chicago; EDWARD J. JAHNKE, JR., Major, U.S.A., M.D., Washington, D. C.

Cardiac Trauma

Moderator: DANIEL C. ELKIN, M.D., F.A.C.S., Atlanta.
Trauma Due to Cardiac Hypoxia. JULIAN JOHNSON, M.D., F.A.C.S., Philadelphia; WILLIAM O. MCQUISTON, M.D., Peoria.
Diagnosis and Treatment of Acute Tamponade. MARK M. RAVITCH, M.D., F.A.C.S., New York.
The Care of Stab and Gunshot Wounds of the Heart. DUANE CARR, M.D., F.A.C.S., Memphis.

Thursday, 8:30—11:30 a.m., Kimball Hall, 306 South Wabash Avenue

Surgery for Congenital Anomalies of the Heart and Great Vessels

Moderator: ALFRED BLALOCK, M.D., F.A.C.S., Baltimore.
When Should the Patient with Congenital Heart Disease Be Referred to the Surgeon? BENJAMIN M. GASUL, M.D., Chicago.
Surgery for the Congenital Heart of the Cyanotic Group. RICHARD L. VARCO, M.D., F.A.C.S., Minneapolis.
Problems Concerning Patent Ductus Arteriosus, Coarctation of the Aorta, and the Double Aortic Arch. JOHN C. JONES, M.D., F.A.C.S., Los Angeles.

Surgical Problems Arising from Acquired Heart Disease

Moderator: EGBERT H. FELL, M.D., F.A.C.S., Chicago.
When Should the Patient with Acquired Heart Disease Be Referred to the Surgeon? OGLESBY PAUL, M.D., Chicago.

Mitral Stenosis. DWIGHT E. HARKEN, M.D., F.A.C.S., Boston.
Surgery for Aortic Stenosis and Mitral and Aortic Insufficiency. THOMAS J. E. O'NEILL, M.D., Philadelphia.

The Latest Developments in the Field of Cardiovascular Surgery

Chairman: WILLIS J. POTTS, Chicago.
Surgery of the Peripheral Venous System. ROBERT R. LINTON, M.D., F.A.C.S., Brookline.
Vessel Grafts. CHARLES A. HUFNAGEL, M.D., F.A.C.S., Washington, D.C.
Arterial Bank. JERE W. LORD, M.D., F.A.C.S., New York.
Surgery of the Peripheral Arterial System. ORMAND C. JULIAN, M.D., F.A.C.S., Chicago.
Congenital Lesions of the Heart and Great Vessels. ROBERT E. GROSS, M.D., F.A.C.S., Boston.
Surgery for Acquired Heart Disease. ROBERT P. GLOVER, M.D., Philadelphia.
Hypothermia. WILFRED G. BIGELOW, M.D., Toronto.
Portacaval Shunt in Portal Hypertension. ARTHUR H. BLAKEMORE, M.D., New York.
Heart-Lung Apparatus. JOHN H. GIBBON, JR., M.D., F.A.C.S., Philadelphia.

TRAUMA TO THE ABDOMEN

JAMES K. STACK, M.D., F.A.C.S., *Chairman*

Tuesday, 8:30—11:30 a.m., John B. Murphy Memorial Auditorium

Moderator: FRANCIS D. MOORE, M.D., F.A.C.S., Boston.
Collaborators:
The Systematic Effects of Trauma. FRANCIS D. MOORE.
Structural Vulnerabilities and Their Relationship to Abdominal Trauma. BARRY J. ANSON, Ph.D. (Med.Sc.), Chicago.
Abdominal Wounds in Korea. JOHN M. OLNEY, M.D., Boston.
Burns and Wounds of the Abdominal Casing. HARVEY S. ALLEN, M.D., F.A.C.S., Chicago.
Post-Traumatic Renal Insufficiency. L. HOLLINGSWORTH SMITH, JR., M.D., Boston.

Wednesday, 8:30—11:30 a.m., John B. Murphy Memorial Auditorium

Moderator: FRANK GLENN, M.D., F.A.C.S., New York.
Collaborators:
Criteria for Treatment Priorities in Multiple Injuries. FRANK GLENN.
Wounds of the Diaphragm, Stomach and Duodenum. CHAMP LYONS, M.D., F.A.C.S., Birmingham.
Wounds of the Small Bowel and Its Mesentery. J. DEWEY BISGARD, M.D., F.A.C.S., Omaha.
Injuries of the Large Bowel, Rectum, and Perineum. R. ARNOLD GRISWOLD, M.D., F.A.C.S., Louisville.
Fractures of the Lumbar Spine and Pelvic Ring as They Relate to the Picture of Abdominal Trauma. THOMAS B. QUIGLEY, M.D., F.A.C.S., Boston.

Thursday, 8:30—11:30 a.m., John B. Murphy Memorial Auditorium

Moderator: CHARLES B. PUESTOW, M.D., F.A.C.S., Chicago.
Collaborators:
Wounds of the Spleen and Its Vessels. AMBROSE H. STORCK, M.D., F.A.C.S., New Orleans.
Wounds of the Liver, Its Ducts, and Its Vessels. I. S. RAVDIN, M.D., F.A.C.S., Philadelphia.
Trauma and Its Impact on Pre-existing Abdominal Lesions. JOHN H. MULHOLLAND, M.D., F.A.C.S., New York.
A Forensic Pathologist's Experience with Abdominal Injuries. JERRY J. KEARNS, M.D., Chicago.

Friday, 8:30—11:30 a.m., John B. Murphy Memorial Auditorium

Moderator: CHARLES G. JOHNSTON, M.D., F.A.C.S., Detroit.
Collaborators:
Injuries to the Major Vessels of the Abdomen and Pelvis. CHARLES G. JOHNSTON, M.D., F.A.C.S., Detroit.
Diagnosis and Treatment of Rupture of the Kidney. J. HARTWELL HARRISON, M.D., F.A.C.S., Boston.
Injuries to the Bladder and Urethra. CARLISLE F. SCHROEDER, M.D., F.A.C.S., Detroit.
Injuries to the Female Genital Tract. RALPH A. REIS, M.D., F.A.C.S., Chicago.
Antibiotics in Abdominal Trauma. CHAMP LYONS, M.D., F.A.C.S., Birmingham.

PANEL DISCUSSIONS

GENERAL SURGERY

Monday, 10:30–12:00 noon, Grand Ballroom, Conrad Hilton

HAROLD L. FOSS, M.D., F.A.C.S., Danville, Pennsylvania, *Presiding.*

Management of Gastric Ulcers

Moderator: HOWARD K. GRAY, M.D., F.A.C.S., Rochester, Minnesota.

Collaborators: JOSEPH E. STRODE, M.D., F.A.C.S., Honolulu, Hawaii; CLAUDE E. WELCH, M.D., F.A.C.S., Boston; W. KEITH WELSH, M.D., F.R.C.S. (Eng.), Toronto.

3:30–5:00 p.m.

Carcinoma of the Mouth

Moderator: JAMES BARRETT BROWN, M.D., F.A.C.S., St. Louis.

Collaborators: JOHN B. ERICH, M.D., F.A.C.S., Rochester, Minnesota: WILLIAM F. MACFEE, M.D., F.A.C.S., New York; TOMAS A. WATSON, M.D., Saskatoon.

Tuesday, 1:30–3:00 p.m., Grand Ballroom, Conrad Hilton

Blood Transfusions; Complications and Their Management

Moderator: PAUL I. HOXWORTH, M.D., F.A.C.S., Cincinnati.

Collaborators: J. GARROTT ALLEN, M.D., F.A.C.S., Chicago; DOUGLAS BLAIR KENDRICK, JR., Colonel, U.S.A., M.D., F.A.C.S., West Point; I. S. RAVDIN, M.D., F.A.C.S., Philadelphia; CARL W. WALTER, M.D., F.A.C.S., Boston.

3:30–5:00 p.m.

Polyposis of the Colon

Moderator: PAUL C. MORTON, M.D., F.A.C.S., New York.

Collaborators: BENTLEY P. COLCOCK, M.D., F.A.C.S., Boston; ELSON B. HELWIG, M.D., Washington, D.C.; ROBERT A. SCARBOROUGH, M.D., F.A.C.S., San Francisco.

Wednesday, 3:30–5:00 p.m., Grand Ballroom, Conrad Hilton

Surgery of the Spleen

Moderator: LOUIS M. ROUSSELOT, M.D., F.A.C.S., New York.

Collaborators: ALEXANDER BLAIN III, M.D., F.A.C.S., Detroit; J. ENGLEBERT DUNPHY, M.D., F.A.C.S., Boston; ROBERT H. E. ELLIOTT, M.D., F.A.C.S., New York.

Thursday, 1:30–3:00 p.m., Grand Ballroom, Conrad Hilton

Malignant Thyroid Disease

Moderator: OLIVER COPE, M.D., F.A.C.S., Boston

Collaborators: R. LEE CLARK JR., M.D., F.A.C.S., Houston; BROWN M. DOBYNS, M.D., F.A.C.S., Cleveland; HENRY H. SEARLS, M.D., F.A.C.S., San Francisco.

GYNECOLOGY AND OBSTETRICS

Wednesday, 1:30–3:00 p.m., Eighth Street Theatre

The Physiological Disorders Present in the Toxemias of Pregnancy

Moderator: ROBERT A. ROSS, M.D., F.A.C.S., Chapel Hill, North Carolina.

Collaborators: ALLAN C. BARNES, M.D., F.A.C.S., Columbus; RUDOLPH A. BARTHOLOMEW, M.D., F.A.C.S., Atlanta; WILLIAM J. DIECKMANN, M.D., Chicago.

3:30–5:00 p.m.

Surgical Procedures in the Treatment of Sterility

Moderator: JOHN ROCK, M.D., F.A.C.S., Brookline, Massachusetts

Collaborators: WESLEY T. POMMERENKE, M.D., Rochester, New York; JAMES J. SWENDSON, M.D., F.A.C.S., St. Paul; NORRIS J. HECKEL, M.D., F.A.C.S., Chicago.

Thursday, 1:30–3:00 p.m., Eighth Street Theatre

Management and Problems of Prolonged Labor

Moderator: JOHN H. MOORE, M.D., F.A.C.S., Grand Forks, North Dakota.

Collaborators: GERALD W. GUSTAFSON, M.D., F.A.C.S., Indianapolis; C. PAUL HODGKINSON, M.D., F.A.C.S., Detroit; JOHN B. MONTGOMERY, M.D., F.A.C.S., Philadelphia.

Friday, 1:30–3:00 p.m., Eighth Street Theatre

Surgical Procedures in the Treatment of Special Defects in the Pelvic Floor Supports

Moderator: ALBERT H. ALDRIDGE, M.D., F.A.C.S., New York.

Collaborators: CHRISTOPHER J. DUNCAN, M.D., F.A.C.S., Brookline, Mass.; MORRIS A. GOLDBERGER, M.D., F.A.C.S., New York; THOMAS C. PEIGHTAL, M.D., F.A.C.S., New York.

3:15–4:45 p.m.

The Treatment of Cancer of the Cervix

Moderator: JOE V. MEIGS, M.D., F.A.C.S., Boston.

Collaborators: CONRAD G. COLLINS, M.D., F.A.C.S., New Orleans; JAMES F. NOLAN, M.D., F.A.C.S., Los Angeles; FRANKLIN L. PAYNE, M.D., F.A.C.S., Philadelphia.

SURGICAL SPECIALTIES

NEUROLOGIC SURGERY

Friday, 1:30–3:30 p.m., West Ballroom, Conrad Hilton

The Neurosurgical Treatment of Epilepsy

Moderator: A. EARL WALKER, M.D., F.A.C.S., Baltimore.

Collaborators: JOHN R. GREEN, M.D., Phoenix; RUSSELL MEYERS, M.D., F.A.C.S., Iowa City; THEODORE B. RASMUSSEN, M.D., Chicago.

3:15–4:45 p.m.

Cerebral Vascular Surgery

Moderator: JAMES L. POPPEN, M.D., F.A.C.S., Boston.

Collaborators: ELDRIDGE H. CAMPBELL, M.D., F.A.C.S., Albany; WALLACE B. HAMBY, M.D., F.A.C.S., Buffalo; CARL F. LIST, M.D., F.A.C.S., Grand Rapids; WILLIAM H. SWEET, M.D., F.A.C.S., Boston.

ORTHOPEDIC SURGERY

Friday, 1:30–3:00 p.m., Gold Room, Congress Hotel

Diagnosis and Treatment of Osteogenic Sarcoma

Moderator: MURRAY M. COPELAND, M.D., F.A.C.S., Washington, D.C.

Collaborators: GRANVILLE A. BENNETT, M.D., Chicago; NORMAN L. HIGINBOTHAM, M.D., F.A.C.S., New York; H. HERMAN YOUNG, M.D., F.A.C.S., Rochester, Minnesota.

3:15–4:45 p.m.

Benign Bone Lesions Sometimes Mistaken for Sarcoma

Moderator: PAUL C. COLONNA, M.D., F.A.C.S., Philadelphia.

Collaborators: G. EDMUND HAGGART, M.D., F.A.C.S., Boston; HERMAN F. JOHNSON, M.D., F.A.C.S. Omaha; MARY S. SHERMAN, M.D., F.A.C.S., New Orleans

PLASTIC SURGERY

Friday, 1:30–3:00 p.m., South Ballroom, Conrad Hilton

Management of Compound Facial Injuries

Moderator: MINOT P. FRYER, M.D., F.A.C.S., St. Louis.

Collaborators: ALBERT D. DAVIS, M.D., F.A.C.S., San Francisco; W. BOWDOIN DAVIS, M.D., F.A.C.S., Baltimore; HANS MAY, M.D., F.A.C.S., Philadelphia.

3:15–4:45 p.m.

Reconstructive Surgery of the Hand

Moderator: WALTER C. GRAHAM, M.D., F.A.C.S., Santa Barbara, California.

Collaborators: HERBERT CONWAY, M.D., F.A.C.S., New York; JOHN TENERY, M.D., Phoenixville, Pennsylvania; FORREST YOUNG, M.D., F.A.C.S., Redland, California.

THORACIC SURGERY

Friday, 1:30–3:00 p.m., John B. Murphy Memorial Auditorium

Thoracic Emergencies

Moderator: WILLIAM M. TUTTLE, M.D., Detroit.

Collaborators: THOMAS H. BURFORD, M.D., F.A.C.S., St. Louis; PAUL W. SANGER, M.D., F.A.C.S., Charlotte, North Carolina; HOWARD E. SNYDER, M.D., F.A.C.S., Winfield, Kansas.

3:15–4:45 p.m.

Postoperative Problems in Thoracic Surgery

Moderator: PAUL C. SAMSON, M.D., F.A.C.S., Oakland.

Collaborators: JOHN C. JONES, M.D., F.A.C.S., Los Angeles; JOHN D. STEELE, M.D., F.A.C.S., Milwaukee; NORMAN J. WILSON, M.D., F.A.C.S., Brookline, Massachusetts.

UROLOGY

Friday, 1:30–3:00 p.m., Upper Tower, Conrad Hilton

Urological Endocrinology

Moderator: CLYDE L. DEMING, M.D., F.A.C.S., New Haven, Connecticut.

Collaborators: WILLIAM P. HERBST, M.D., F.A.C.S., Washington, D.C.; THOMAS D. MOORE, M.D., F.A.C.S., Memphis; SAMUEL A. VEST, M.D., F.A.C.S., Charlottesville.

3:15–4:45 p.m.

Carcinoma of the Prostate; an Appraisal of the Present-Day Methods of Treatment

Moderator: J. HARTWELL HARRISON, M.D., F.A.C.S., Boston.

Collaborators: ROGER BAKER, M.D., Chicago; GRAYSON CARROLL, M.D., F.A.C.S., St. Louis; RUBIN FLOCKS, M.D., F.A.C.S., Iowa City; REED M. NESBIT, M.D., F.A.C.S., Ann Arbor; PARKE G. SMITH, M.D., F.A.C.S., Miami.

OPHTHALMOLOGY

Tuesday, 8:30–10:00 p.m., North Ballroom, Conrad Hilton

Surgery of the Eyelids and Adnexae

Moderator: MERRILL J. REEH, M.D., F.A.C.S., Oregon.

Collaborators: J. GORDON COLE, M.D., F.A.C.S., New York; CHARLES E. ILIFF, M.D., F.A.C.S., Baltimore; BYRON SMITH, M.D., New York.

Wednesday, 8:30–10:00 p.m., North Ballroom, Conrad Hilton

Surgical Management of Glaucoma

Moderator: SAMUEL J. MEYER, M.D., F.A.C.S., Chicago.

Collaborators: A. GERARD DE VOE, M.D., F.A.C.S., New York; P. ROBB MCDONALD, M.D., F.A.C.S., Philadelphia; WILLIS S. KNIGHTON, M.D., F.A.C.S., New York.

Thursday, 8:30–10:00 p.m., North Ballroom, Conrad Hilton

Surgical Management of Cataracts

Moderator: ERLING W. HANSEN, M.D., F.A.C.S., Minneapolis.

Collaborators: FREDERICK C. CORDES, M.D., F.A.C.S., San Francisco; JOHN M. MCLEAN, M.D., New York; ALBERT D. RUEDEMANN, M.D., F.A.C.S., Detroit.

OTOLARYNGOLOGY

Tuesday, 8:30–10:00 p.m., South Ballroom, Conrad Hilton

The Tonsil and Adenoid Problem

Moderator: GORDON F. HARKNESS, M.D., F.A.C.S., Davenport.

Collaborators: HEYWORTH N. SANFORD, M.D., Chicago; FREDERICK T. HILL, M.D., F.A.C.S., Waterville, Maine; DEAN M. LIERLE, M.D., F.A.C.S., Iowa City; C. STEWART NACH, M.D., F.A.C.S., Rochester, New York.

Wednesday, 8:30–10:00 p.m., South Ballroom, Conrad Hilton

Reconstructive Surgery of the Ear and Nose

Moderator: SAMUEL SALINGER, M.D., F.A.C.S., Chicago.

Collaborators: LAWRENCE R. BOIES, M.D., Minneapolis; FRED Z. HAVENS, M.D., F.A.C.S., Rochester, Minnesota; JEROME P. WEBSTER, M.D., F.A.C.S., New York.

Thursday, 8:30–10:00 p.m., South Ballroom, Conrad Hilton

Menière's Syndrome

Moderator: FREDERICK T. HILL, M.D., F.A.C.S., Waterville, Maine.

Collaborators: GORDON D. HOOPLE, M.D., Syracuse; FRANK D. LATHROP, M.D., F.A.C.S., Boston; THEODORE E. WALSH, M.D., St. Louis.

FORUM ON FUNDAMENTAL SURGICAL PROBLEMS

Heart

Monday, 9:00 a.m.—12:00 noon, North Ball Room, Conrad Hilton Hotel

Studies on Techniques Permitting Prolonged Cardiac Venous Inflow Stasis. ERNEST M. BERKAS, DONALD J. FERGUSON, JOSEPH J. GARAMELLA, and RICHARD L. VARCO, Minneapolis.

Autogenous Lung Oxygenator with Total Cardiac By-pass for Intracardiac Surgery. MORLEY COHEN and C. WALTON LILLEHEI, Minneapolis.

A Study of Hibernation in Relation to the Technique of Hypothermia for Intracardiac Surgery. G. A. TRUSLER, J. E. MCBIRNIE, F. G. PEARSON, A. G. GORNALL, and W. G. BIGELOW, Toronto.

Studies of Circulation during Hypothermia. SAMUEL KAPLAN, JAMES A. HELMSWORTH, LELAND C. CLARK, JR., and ROGER T. SHERMAN, Cincinnati.

Studies on Occlusion of the Inferior Vena Cava Above and Below the Hepatic Veins, in Normothermic and Hypothermic Dogs. J. CUTHBERT OWENS, EDWARD B. LIDDLE, LEON TUNE, and IRVIN ZEAVIN, Denver.

Production and Repair of Experimental Interventricular Septal Defects Under Direct Vision with the Aid of Hypothermia. M. TAUFIC and F. JOHN LEWIS, Minneapolis.

Digital Exploration of the Ventricular Chambers of the Heart Through a Rubber Diverticulum; the Repair of Ventricular Septal Defects. WILLIAM W. L. GLENN, WILLIAM E. BLOOMER, THOMAS O. GENTSCH, and HAYES H. CLUXTON, New Haven.

The Use of a Vent for the Left Ventricle as a Means of Avoiding Air Embolism to the Systemic Circulation During Open Cardiotomy with the Maintenance of the Cardiorespiratory Function of Animals by a Pump Oxygenator. BERNARD J. MILLER, JOHN H. GIBBON, JR., VICTOR F. GRECO, C. HAROLD COHN, and FRANK F. ALLBRITTEN, JR., Philadelphia.

The Blue Dye Method for Cardiac Output; a Practical Technique with Clinical Results. FRANK C. WILSON, JR., WILLIAM A. NEELY, and JAMES D. HARDY, Memphis.

Polarographic Measurement of Oxygen Tension in Whole Blood and Tissues During Total By-pass of the Heart. LELAND C. CLARK, JR., JAMES A. HELMSWORTH, SAMUEL KAPLAN, ROGER T. SHERMAN, and ZENA TAYLOR, Cincinnati.

Healing Patterns Following Closure of Experimental Defects of the Atrial Septum. E. WATKINS, A. A. POMERANZ, and ROBERT E. GROSS, Boston.

Methods of Closure of Experimentally-Produced Atrial Septal Defects. DAVID E. DONALD, JOHN W. KIRKLIN, F. HENRY ELLIS, JR., and JOHN H. GRINDLAY, Rochester, Minnesota.

The Effect of Interatrial Septal Defect Shunts upon Susceptibility to Bacterial Endocarditis. ROBERT N. HAMMERSTROM, MITCHELL W. SPELLMAN, MORLEY COHEN, and C. WALTON LILLEHEI, Minneapolis.

Lungs and Peripheral Circulation

Monday, 9:00 a.m.—12:00 noon, 8th Street Theatre

Experimental Production of Cyanosis in Dogs by a One-Stage Operation. E. WATKINS, A. A. POMERANZ, E. I. GOLDSMITH, and ROBERT E. GROSS, Boston.

Experimental Bronchial Transection, Resection, and Transplantation in the Growing Pig. LAWRENCE B. KIRILUK, and K. ALVIN MERENDINO, Seattle.

Use of a Venturi Meter for Clinical Measurement of Maximal Breathing Capacity. DAVID Y. COOPER, W. S. BLAKEMORE, JULIAN JOHNSON, and JONATHAN E. RHOADS, Philadelphia.

Circulatory Factors Contributing to Alterations in Pulmonary Vascular Histology. DONALD J. FERGUSON, ERNEST M. BERKAS, and RICHARD L. VARCO, Minneapolis.

The Effect of Experimental Lung Ischemia on Pulmonary Function. BRIAN BLADES, HOWARD PIERPONT, ABDUSSAMED SAMADI, and ROBERT P. HILL, Washington.

Studies on Pulmonary Function Utilizing the Method of Controlled Unilateral Bronchovascular Occlusion—I, Main Pulmonary Artery Pressure Response to Occlusion and to the Valsalva Maneuver. PAUL NEMIR, JR., H. H. STONE, T. N. MACKRELL, and H. R. HAWTHORNE, Philadelphia.

An Experimental Study of the Effects of the Open Chest, Pneumoperitoneum, Resistance to Expiration and Variation of Tidal Volume in the Anesthetized Dog. ROGER H. L. WILSON, VICTOR RICHARDS, and RICHARD S. HAHN, San Francisco.

Physiological Changes Attending Bilateral Pulmonary Resection. ARTHUR A. SIEBENS, RALPH E. SMITH, GRAFTON A. SMITH, and CLIFFORD F. STOREY, St. Albans, New York.

The Experimental Production of Peripheral Arterial Disease—I, Large Scale Remote Control Chronic Stimulation. RUDOLPH A. LIGHT, CARL R. ZIMMER, and JOHN H. DEWITT, JR., Nashville.

An Evaluation of Treatment of Essential Hypertension by Combined Adrenalectomy and Sympathectomy. H. A. ZINTEL and J. A. MACKIE, Philadelphia.

An Explanation for Some Local Complications Produced by Intraarterially Administered Thorotrast. EBERHARD TRAMS, CALVIN T. KLOPP, and BRIAN BLADES, Washington.

A Closed Procedure for Red Cell Volume and Red Cell Turnover Studies in Man Using Cr 51. RAYMOND C. READ, Boston.

The Method for the Sterilization and Preservation of Arterial Homografts and Heterografts. CHARLES A. HUFNAGEL, Washington.

Selective Angiocardiography. JOHN F. NEVILLE, JR., BERNARD BERCU, and THOMAS H. BURFORD, St. Louis.

Burns, Wounds, and Anesthesia

Monday, 2:00—5:00 p.m., North Ball Room, Conrad Hilton Hotel

Penetration of Heat in Thermal Burns. PHILIP B. PRICE, FARREL L. HANSEN, and DEAN CALL, Salt Lake City.

Radioactive Techniques in the Study of Protein Metabolism of Severe Burn Patients—I, Studies with Radioactive Iodinated Human Serum Albumin. TRUMAN G. BLOCKER, JR., WILLIAM C. LEVIN, C. C. SNYDER, S. R. LEWIS, and WILLIAM R. HURST, Galveston.

Changes in Tissue Sodium and Potassium of Rats Following a Severe Burn. JAMES M. WALKER, MARIE T. BARNES, AVIS R. WALKER, and ELIZABETH M. BARNETT, Philadelphia.

The Use of Proteolytic Enzymes in the Débridement of Burn Eschar. LOUIS M. ROUSSELOT and JAMES F. CONNELL, JR., New York.

A Study on Cross Resistance of Bacteria from Burn Wounds to Aureomycin, Chloramphenicol, and Terramycin. W. CHANDLER THOMPSON, JR., Richmond, Virginia.

Distribution of Antibiotics in Blood and Tissues with Particular Reference to Penetration through Skin, Fat, and Fascia. CHESTER W. HOWE, WILLIAM C. WIGGLESWORTH, and ROBERT E. KAHN, Boston.

Further Observations on the Use of Phisohex in the Preoperative Scrub; Relative Effectiveness After One Year; Use of Brushes; and Use of Antiseptic Hand Dip. HAROLD A. ZINTEL and HELEN ELLIS, Philadelphia.

Intraperitoneal Administration of Terramycin in the Treatment of Acute Bacterial Peritonitis. WILLIAM E. SCHATTEN, JOHN D. DESPREZ, TED N. CLINE, and WILLIAM E. ABBOTT, Cleveland.

Utilization of Stainless Steel Through-and-Through Sutures and Wound Splints in the Closure of Complicated Abdominal Wall Wounds. CLARENCE DENNIS, CARLTON A. NELSON, and FRANK B. ANKNER, Brooklyn.

Effect of Cyclopropane Anesthesia on Pulmonary Artery Pressure in Humans. BENJAMIN ETSTEN, HAROLD F. RHEINLANDER and ROBERT N. REYNOLDS, Boston.

Oxygen Consumption of Tissues after Anesthesia at Low Temperature. WALTER J. BURDETTE, New Orleans.

A Controlled Study of the Effectiveness and Complications of Long-Lasting Intercostal Nerve Block for Major Upper Abdominal Surgery. EDWARD B. MCKENZIE and DOUGLAS W. EASTWOOD, St. Louis.

Obstetrics and Gynecology

Monday, 2:00—5:00 p.m., 8th Street Theatre

The Peripheral Circulation in Normal Pregnancy. CARL M. HERBERT, EDWARD A. BANNER, and KHALIL G. WAKIM, Rochester, Minnesota.

Maternal and Fetal Blood Estimation Following the Administration of Chloral Hydrate During Labor. J. BERNARD BERNSTINE, ARTHUR E. MEYER, and HARRY B. HAYMAN, Philadelphia.

Isotope Tracer Studies on the Mechanism of Amniotic Fluid Formation; Preliminary Report. ALBERT A. PLENTL and DONALD L. HUTCHINSON, New York.

Some Hormonal Aspects of Human Delivery with Particular Reference to the Neurohypophysis. ROBERT C. HICKEY and WILLIAM C. KEETTEL, Iowa City.

Fibrinolysin Titers as Affected by Labor and Delivery. R. R. MARGULIS, JOHN H. LUZADRE, and C. PAUL HODGKINSON, Detroit.

The Clinical Pattern of Adenomatous Hyperplasia and Its Pathologic Background. SAUL B. GUSBERG, New York.

Effective Thyroid Medication on Menstrual Disorders and Sterility. C. L. BUXTON, New York.

Surgery or Radiotherapy—A Test for the Selection of Treatment in Cancer of the Cervix. JOHN B. GRAHAM, RUTH M. GRAHAM, and WINIFRED LIU, Boston.

Heart and Great Vessels

Tuesday, 9:00 a.m.—12:00 noon, North Ball Room, Conrad Hilton Hotel

The Surgical Correction of Mitral Insufficiency. DWIGHT E. HARKEN, HARRISON BLACK, LEWIS DEXTER, and LAURENCE B. ELLIS, Boston.

The Clinical Significance of Direct Measurement of Left Atrial and Pulmonary Artery Pressures Before and After Mitral Valvotomy. RUSSEL H. LEE, GEORGE ORDWAY, and FRANK GERBODE, San Francisco.

Immediate Hemodynamic Effects of Mitral Valvotomy During Surgery for Mitral Stenosis. RALPH LEV, JOHN W. KIRKLIN, DANIEL C. CONNOLLY, and EARL H. WOOD, Rochester, Minnesota.

Observations on Sodium Balance in Patients Undergoing Mitral Valvotomy. QUENTIN DEMING and FRANK GERBODE, San Francisco.

Acute and Chronic Water and Electrolyte Disturbances Seen in the Surgical Management of Mitral Stenosis. GRAHAM WILSON, LAURA BROOKS, ALLAN MYRDEN, DWIGHT E. HARKEN, and FRANCIS D. MOORE, Boston.

Cardiodynamics of Experimental Septal Defects in Dogs. HAROLD KING, WARREN COGGESHALL, and PAUL R. LURIE, Indianapolis.

The Experimental Production of Tricuspid Insufficiency and its Relation to Ascites. JOSEPH L. SPRAFKA, FRANCIS J. HADDY, JOHN F. ALDEN, and IVAN D. BARONOFSKY, St. Paul.

Study of Prosthetic Materials Used Inside the Heart. CLARE B. BAKER and HENRY T. BAHNSON, Baltimore.

Further Studies of the Renal Factor in the Hypertension of Experimental Coarctation of the Aorta. H. A. COLLINS, A. M. LANGA, N. S. OLSEN, and H. W. SCOTT, JR., Nashville.

A Follow-Up Report on the Development of a Plastic Prosthesis for the Atrioventricular Valve. G. REHMI DENTON, THOMAS SEYMOUR, and HAROLD WIGGERS, Albany.

Factors in the Production and Treatment of Experimental Cardiac Tamponade Using a Standard Wounding Technique. H. LEROY BROCKMAN, DENTON A. COOLEY, and RUSSELL A. HUGGINS, Houston.

Use of Quinidine in Fibrillation and Defibrillation of the Canine Heart. LOUIE B. JENKINS, FRED E. NIGELS, and JOHN A. BOONE, Charleston, South Carolina.

Coronary Circulation During Ventricular Fibrillation; an Experimental Study. JAMES A. HELMSWORTH, LELAND C. CLARK, JR., SAMUEL KAPLAN, and ROGER T. SHERMAN, Cincinnati.

Ventricular Fibrillation; an Experimental Study. C. K. KIRBY, JULIAN JOHNSON, J. ENGELBERG, and R. ROVIS, Philadelphia.

Pancreas, Liver and Intestine

Tuesday, 9:00 a.m.—12:00 noon, Eighth Street Theatre

Pancreatic Duct Pressure Studies in Dogs With and Without Occlusion of the Duct System. RICHARD H. EGDAHL, EDWIN L. BRACKNEY, and WILLIAM C. KELLY, Minneapolis.

Further Observations on the Role of Bile in the Etiology of Acute Hemorrhagic Pancreatitis. BERNARD FISHER and EDWIN R. FISHER, Pittsburgh.

Plasma Electrolyte Studies Following Experimental Fat Necrosis. M. B. WHEAT and ROBERT ELMAN, St. Louis.

Venous Thrombosis as a Precipitating Factor in Acute Necrotizing Hemorrhagic Pancreatitis. THOMAS W. ADAMS and MERLE M. MUSSELMAN, Eloise, Michigan.

The Electrolyte Pattern Following Total Hepatectomy. BERNARD FISHER, FRANK M. MATEER, CLEM RUSS, and HERBERT URAM, Pittsburgh.

The Intrahepatic Circulation in the Normal and in the Diseased Liver. JOHN L. MADDEN, JOHN M. LORÉ, JR., FRANK P. GEROLD, and JACOB M. RAVID, New York.

Rearterialization of Rat Liver After Interruption of Hepatic Artery. ROBERT P. DELONG, Philadelphia.

Experimental Studies on Hepatic Venous Pressure in Relation to Certain Alterations of Hepatic Circulation. PAUL L. OGBURN, JOHN H. GRINDLAY, and JESSE L. BOLLMAN, Rochester, Minnesota.

The Effect of Portacaval Shunts on Liver Blood Flow. GEORGE L. NARDI, WILLIAM V. MCDERMOTT, JR., and ERNEST DOBSON, Boston.

Late Follow-Up on Experimental Hepatic-Portal Arteriovenous Fistulae. J. A. SCHILLING and F. W. MCKEE, Rochester, New York.

Massive Intestinal Resection in Experimental Ascites. VICTOR M. BERNHARD, ROBERT MONK, W. HARRISON MEHN, and HAROLD LAUFMAN, Chicago.

The Effect of Insulin Hypoglycemia upon Distal Colon Motility. IRVING F. STEIN, JR., EDWARD GOLDBERG, and KARL A. MEYER, Chicago.

A New Method of Producing Duodenal and Gastric Ulcers in Dogs. EDWARD B. C. KEEFER, FRANK GLENN, and KIRBY A. MARTIN, New York.

Cancer and Endocrine Glands

Tuesday, 2:00—5:00 p.m., North Ball Room, Conrad Hilton Hotel

Carcinogenic Effect of Imbedding Various Plastic Films in Rats and Mice. B. S. OPPENHEIMER, ENID T. OPPENHEIMER, and ARTHUR PURDY STOUT, New York.

Mechanism of Regression of Mammary Cancers after Adrenalectomy. THOMAS L. DAO, Chicago.

The Urinary Steroids in Adrenalectomized, Ovariectomized Women with Mammary Carcinoma. BERNARD ZIMMERMANN, HENRY S. BLOCH, and BETTY J. OSEID, Minneapolis.

Uterine Nucleic Acid Synthesis as a Function of Steroid Hormone Levels. N. I. GOLD and S. H. STURGIS, Boston.

The Use of a β Ray Point Source for Destruction of the Hypophysis. T. B. RASMUSSEN and PAUL V. HARPER, JR., Chicago.

Effects of Hypophysectomy on Malignant Tumor Induction by 2-Acetyl-Amino-Fluorene. STANLEY C. SKORYNA and D. R. WEBSTER, Montreal.

Disappearance Rates of Na^{24}, K^{42}, P^{32}, and I^{131} From Cerebral Cortex and Muscle in the Dog. WALTER LAWRENCE and LEON HELLMAN, New York.

Proposed Method of Greatly Increasing Roentgen Dosage in Susceptible Tumors of Childhood; Experimental Use of Lead Shields to Spleen. WINFIELD L. BUTSCH, G. N. SCATCHARD, S. ANTHONE, R. ANTHONE, and J. B. Dumm, Buffalo.

The Prevention of Peritoneal Tumor Implants by Radioactive Colloidal Gold. EDWARD R. WOODWARD, and PAUL V. HARPER, JR., Chicago.

Clinical Experience with a New Method for Estimating Serum Acid Phosphatase of Prostatic Origin. FREDDY HOMBURGER and W. H. FISHMAN, Boston.

Development of Both Thyroid Carcinoma and Pituitary Tumors in Mice Fed Propylthiouracil. GEORGE E. MOORE, FREDERICK G. BOCH, Buffalo, and EDWIN L. BRACKNEY, Minneapolis.

Cholesterol and Cholic Acid Excretion in the Bile of Hyper-, Hypo- and Euthyroid Rats. JAMES C. THOMPSON and HARRY M. VARS, Philadelphia.

Studies on Autotransplantation of Thyroid and Adrenal Glands in Dogs. E. B. LIDDLE, G. WITTENSTEIN and HENRY SWAN, Denver.

Plastic Surgery

Tuesday, 2:00—5:00 p.m., Upper Tower, Conrad Hilton Hotel

Development of Circulation in Homografts of Skin and Observations on the Effect of an Antihistaminic (Benadryl) and of Burying Homotransplants of Skin. HERBERT CONWAY, RICHARD B. STARK, DOYLE JOSLIN, and ANTHONY JEROME, New York.

Effect of X-Ray Irradiation on Skin Capillary Clearance. R. GEE, M. T. EDGERTON, JR., and F. S. HOFFMEISTER, Baltimore.

Postmortem Homografts as Biological Dressings for Extensive Burns. JAMES BARRETT BROWN, MINOT P. FRYER, MILTON LU, and PETER RANDALL, St. Louis.

Comparison of the Healing of Experimental Burns under "Pressure" Dressing Vs. "Open" Treatment. E. S. SHAYA, F. S. HOFFMEISTER, S. P. HOLMAN, and M. T. EDGERTON, JR., BALTIMORE.

Factors Influencing the Survival of Successful Skin Homografts in the Chicken—I, Effects of Varying Age of Donor and Recipient. JACK A. CANNON, ROBERT A. WEBER, and W. P. LONGMIRE, JR., Los Angeles.

Further Studies on the Effects of Various Agents on Healing in Controlled Partial-Thickness Skin Loss. PETER RANDALL, HERMAN NEWMAN, CARLOS FERRATTI, and JAMES BARRETT BROWN, St. Louis.

A Quantitative Method for Timing Safe Transfer of Skin Pedicle Flaps. E. S. SHAYA, M. T. EDGERTON, JR., and F. S. HOFFMEISTER, Baltimore.

Investigation of the Use of Polyvinyl Compounds as Subcutaneous Prostheses. JAMES BARRETT BROWN, MINOT P. FRYER, MILTON LU, and ANDREW M. MOORE, St. Louis.

Shock and Plasma Expanders

Wednesday, 9:00 a.m.—12:00 noon, North Ball Room, Conrad Hilton Hotel

The Effect of Variation in Hepatic Blood Flow on the Course of Shock. MURRAY N. ANDERSEN, JOHN D. STEWART, ROGER S. HUBBARD, WILLIAM H. POTTER, and JAMES M. GARVEY, Buffalo.

Studies on the Role of Bacteria in Irreversible Hemorrhagic Shock in Dogs. ERIC G. HARDY, GEORGE C. MORRIS, JR., ELLARD M. YOW, B. W. HAYNES, JR., and MICHAEL E. DEBAKEY, Houston.

Blood Ammonia in Normal and Shock States. RUSSELL M. NELSON and DAVID SELIGSON, Washington.

A Comparison of Intra-Arterial and Intravenous Transfusions in Treatment of Hemorrhagic Shock. RALPH C. RICHARDS and FARREL L. HANSEN, Salt Lake City.

A Controlled Comparison of Intra-Arterial and Intravenous Transfusion: Effect on Blood Pooling and Survival Rate in Experimental Hemorrhagic Shock. JAMES V. MALONEY, JR., CHEVES MCC. SMYTHE, JOSEPH P. GILMORE, and STANLEY W. HANDFORD, Camp Lejeune, North Carolina.

Cerebral Hemodynamics in Controlled Hypotension. GEORGE C. MORRIS, JR., JOHN H. MOYER, HARVEY B. SNYDER, and B. W. HAYNES, JR., Houston.

Similarity of Cerebral Blood Flow, Arterial Pressure and Cardiac Rate Responses to Experimental Cerebral Concussion and Electroconvulsive Shock. GEORGE BROWN and RUSSELL MEYERS, Iowa City.

Evaluation of Blood Volume Determinations by Radioactive Iodinized Albumin in Severe Hemorrhage. VINTON E. SILER, Cincinnati.

Clinical Experience with Plasma Augmenters, Dextran, and PVP. P. G. WEIL and D. R. WEBSTER, Montreal.

Study of the Effect of the Administration of Dextran and Physiological Saline Solution on the Colloidal Osmotic Pressure of the Blood in Splenectomized Dogs Following Hemorrhage. CHARLES ECKERT, T. E. WEICHSELBAUM, and RUTH SIGHTS, St. Louis.

The Effect of Polyvinylpyrrolidone Retention on Liver Pathology and Physiology. WILLIAM A. ALTEMEIER, LEON SCHIFF, EDWARD A. GALL, DAVID HAMILTON, and JEROME GIUSEFFI, Cincinnati.

The Determinants of the Efficacy of Various Expanders in Plasma Volume Expansion and Maintenance in Normal Subjects. WILLIAM METCALF, LOUIS M. ROUSSELOT, FRANK E. GILBERTSON, and JAMES M. HARMON, New York.

A Clinical Method for the Determination of the Molecular Weights and the Molecular Weight Distributions of Plasma Expanders. WILLIAM METCALF, LOUIS M. ROUSSELOT, FRANK E. GILBERTSON, and JAMES M. HARMON, New York.

Changes in Adrenocortical Steroid Concentration of Peripheral Plasma Following Surgery. J. A. MONCRIEF, T. E. WEICHSELBAUM, and ROBERT ELMAN, St. Louis.

Gastrointestinal Tract

Wednesday, 9:00 a.m.—12:00 noon, 8th Street Theatre

A Comparative Evaluation of Tubular Gastric Resection. LLOYD D. MACLEAN, Minneapolis.

Gastrojejunostomy and Finney Pyloroplasty; Their Effects upon Heidenhain Pouch Secretion in Vagotomized and Nonvagotomized Dogs. LLOYD M. NYHUS, EDMUND A. KANAR, HORACE G. MOORE, JR., LESTER R. SAUVAGE, EVERETT J. SCHMITZ, EDWARD H. STORER, and HENRY N. HARKINS, Seattle.

Anatomic and Physiologic Reconstruction Following Total Gastrectomy by the Use of a Jejunal Food Pouch. ROBERT P. HAYS, McKinney, Texas.

Esophageal Replacement by Use of the Right Colon, a One Stage Thoracoabdominal Procedure; an Experimental and Clinical Study. J. STANLEY BATTERSBY, Indianapolis.

The Role of Potassium in the Pathogenesis and Treatment of the Postgastrectomy Dumping Syndrome. ALLEN KLEIMAN and AUSTIN R. GRANT, Phoenix.

Acute Alterations in Blood Volume, Plasma Electrolytes and the Electrocardiogram Produced by Oral Administration of Hypertonic Solutions to Totally Gastrectomized Patients. KATHLEEN E. ROBERTS, HENRY T. RANDALL, and HOLLON W. FARR, New York.

Effect of Vagus Section and Isolation of the Pyloric Antrum on the Development of the Mann-Williamson Ulcer in the Dog. JAMES OLIVER, Chicago.

The Importance of the Spatial Relationship of the Antrum in the Development of Gastrojejunal Ulcer in the Dog. W. D. KELLY and F. S. CROSS, Minneapolis.

Observations upon Peptic Activity of the Gastric Contents in Normal Individuals and in Patients with Peptic Ulceration. DOUGLAS A. FARMER, PAUL M. BURKE, and REGINALD H. SMITHWICK, Boston.

The Importance of Deficient External Pancreatic Secretion During Banthine Treatment of Duodenal Peptic Ulcer. R. M. WHITROCK, R. W. HANSEN, and M. B. CHENOWETH, Ann Arbor.

Partition of Humoral and Vagal Stimulated Gastric Secretion in Human Beings. ARTHUR M. SCHOEN and R. ARNOLD GRISWOLD, Louisville.

Strangulation Obstruction—IV, Studies on the Relationship Between Bacterial Content, Spectrophotometric Abnormality, and Toxicity of Injected Fluids. PAUL NEMIR, JR., H. R. HAWTHORNE, and D. L. DRABKIN, Philadelphia.

Strangulation Obstruction—Antibiotic Protection Study; Preliminary Report. ISIDORE COHN, JR., New Orleans.

Observation on Splanchnic and Hepatic Blood Flow in Experimental Acute Small Intestinal Obstruction. ROBERT WATMAN, JACK THOMPSON, JOSEPH MORTON, and EDWIN ELLISON, Columbus, Ohio.

Urology

Wednesday, 9:00 a.m.—12:00 noon, South Ball Room, Conrad Hilton Hotel

Changes in the Renal Collecting System, Studied by Intravenous Pyelography, Following Ureteral Anastomosis with Isolated Small Intestinal Segments. EUGENE M. BRICKER, HARVEY R. BUTCHER, JR., and ALAN MCAFEE, St. Louis.

A Physiological Study of Vesical Lymphatics. ROGER BAKER, DUNCAN E. GOVAN, and JOHN SAWYER, Chicago.

The Experimental Background for Retroperitoneal Radiation by Radioactive Colloids. FRANK HINMAN, JR., GILBERT I. SMITH, and ELMER NG, San Francisco.

Spontaneous Afibrinogenemia in Cancer of the Prostate. JAY J. CRANE, ARNOLD G. WARE, and JACK HAMILTON, Los Angeles.

The Results of Trigone-Sigmoid Transplant in the Dog. MARK E. SMITH, FRANCIS W. STREHL, C. W. VERMEULEN, and WILLIAM J. GROVE, Chicago.

Vesical Orifice Obstruction in Women and its Treatment by Transurethral Resection. DAVID M. DAVIS, Philadelphia.

Furadantin. W. J. TOLAND and JAMES C. KIMBROUGH, Washington.

Significance of the Prostatic Secretion Acid Phosphatase as a Diagnostic and Prognostic Index in Carcinoma of the Prostate. JABER MUHSEN, Columbus, Ohio.

Nutrition and Electrolytes

Wednesday, 2:00—5:00 p.m., North Ball Room, Conrad Hilton Hotel

The Intestinal Absorption of Amino Acids Following Gastrectomy. RICHARD E. GARDNER, HAROLD A. HARPER, ALLEN H. JOHNSON, FREDERICK M. BINKLEY, QUENTIN BONSER, and HORACE J. MCCORKLE, San Francisco.

Comparative Absorption of Amino Acids from the Intestine. CHARLES G. JOHNSTON and A. U. ORTEN, Detroit.

Tube Feeding with Liquefied Whole Food. JAMES BARRON and LAURENCE S. FALLIS, Detroit.

Thyroid Adrenocortical Interrelationships During Surgical Convalescence. IRA S. GOLDENBERG and MARK A. HAYES, New Haven.

Influence of Nutrition on the Response to Intravenous ACTH. MORTON PAREIRA, W. HICKS, and ROBERT ELMAN, St. Louis.

Recent Studies of Hyperadrenocorticism; Its Pathologic Physiology and Management. J. HARTWELL HARRISON and JOHN C. LAIDLAW, Boston.

A Simplified Method for Clinical Estimation of Acid-Base Balance. ARTHUR COHEN and RUSSELL M. NELSON, Washington.

The Effect of Surgery on the Extracellular Water and Distribution of Electrolytes. HERMAN DELANCY, ALBERT MOWLEM, and BERNARD ZIMMERMANN, Minneapolis.

Studies of Water Distribution in Patients with Laennec's Cirrhosis Using Radioactive Sodium (Na^{24}). HELENA GILDER, F. REDO, and DAVID P. BARR, New York.

Metabolic Alterations in Surgical Patients—II, The Excretion of Salt and Water Postoperatively Following the Administration of Various Sodium Containing Solutions. WILLIAM DRUCKER, HARVEY KRIEGER, LOUIS I. BABB, STANLEY LEVEY, and WILLIAM E. ABBOTT, Cleveland.

The Effect of Hexamethonium Induced Hypotension on Water and Electrolyte Excretion in Man. HARVEY B. SNYDER, GEORGE C. MORRIS, JOHN H. MOYER, and B. W. HAYNES, JR., Houston.

Observations on the Distribution Volume of Sodium Thiosulfate. E. A. BOLING and G. M. WILSON, Boston.

Surgery in the Elderly Patient; the Metabolism of Water. PAUL HODGSON, VIVIAN IOB, and ROBERT BERRY, Ann Arbor.

Metabolic Reaction to Staged Operations in Man; a Preliminary Report. JAMES D. HARDY and FRANCIS H. COLE, Memphis.

Orthopedic Surgery

Wednesday, 2:00—5:00 p.m., Upper Tower, Conrad Hilton Hotel

Histochemical Studies in Bone Diseases—I, Acid and Alkaline Phosphatase Distribution in Bone Tumors as a Diagnostic Aid. W. S. GILMER, Memphis.

The Effect of Compound F Upon Experimental Arthritis in Rabbits. R. H. RAMSEY and J. ALBERT KEY, St. Louis.

Resistant Nontuberculous Infections of Bone; Experimental Use of Iproniazid (Marsilid) in the Treatment. DAVID M. BOSWORTH and J. WILLIAM FIELDING, New York.

Use of Plastic Materials in the Joints of Animals. MILTON C. COBEY, Washington.

A Study on the Effect of Pressure on the Healing of Bone. LEE T. FORD and J. ALBERT KEY, St. Louis.

Irradiation of Bone in the Presence of Intramedullary Metallic Fixation; a Preliminary Report. JACK WICKSTROM, ROBERT H. HUTCHINSON, EDWARD T. HASLAM, and J. V. SCHLOSSER, New Orleans.

Changes in Bone Following Exposure to Ultrasonic Energy. N. I. ARDAN, JR., JOSEPH M. JANES, and J. F. HERRICK, Rochester, Minnesota.

Strength of Intervertebral Discs. F. GAYNOR EVANS and HERBERT R. LISSNER, Detroit.

The Use of Radioactive Phosphorus in Determining the Viability of Femoral Heads in Dogs After Subcapital Fractures. E. BRUCE TOVEE, Toronto.

The Lower Nephron and Blood Coagulation

Thursday, 9:00 a.m.—12:00 noon, North Ball Room, Conrad Hilton Hotel

Peripheral Self-Regulatory Effects of Blood Pressure. ARNOLD H. WILLIAMS, SIGMUND A. WESOLOWSKI, ALBERT EINHEBER, and EDWIN J. PULASKI, Washington.

Early Changes in Ultra Structure of Renal Tubular Epithelium Associated with the Production of Lower Nephron Nephrosis. C. BARBER MUELLER and FRED CALDWELL, St. Louis.

Experimental Renal Tubular Necrosis. KENNETH OWEN, ROBERT DESAUTELS, and CARL W. WALTER, Boston.

Species Difference in Renal Response to Trauma. MICHAEL LADD and EDWIN J. PULASKI, Washington.

The effects of Controlled Hypotension on Glomerular Filtration and Renal Plasma Flow in Man. B. W. HAYNES, JR., GEORGE C. MORRIS, JOHN H. MOYER, and HARVEY B. SNYDER, Houston.

A Simplified Artificial Kidney and Ultrafiltrator. WILLIAM Y. INOUYE and JOSEPH ENGELBERG, Philadelphia.

An Analysis of the Importance of Antithrombin in Thromboembolism. T. D. PEMRICK and MERLE M. MUSSELMAN, Eloise, Michigan.

Experimental Pulmonary Embolism. W. R. CATE, JR., RUDOLPH A. LIGHT, WILLIAM H. EDWARDS, and ROLLIN A. DANIEL, JR., Nashville.

Pulmonary Vasoconstriction Associated with Minute Pulmonary Emboli. JOHN J. BYRNE, RAMON ISALES, and VINCENT J. HICKEY, Boston.

Fibrinolysis in Surgery Patients—II, Normal Fibrinogen-Fibrinolysin Relationships. WILLIAM W. COON and PAUL E. HODGSON, Ann Arbor.

Studies of Intravascular Clot Lysis with Human Fibrinolysin. EUGENE E. CLIFFTON, New York.

The Use of Phosphine 3R, a Water-Soluble Fluorochrome, in the Diagnosis of Fat Embolism. LEONARD F. PELTIER, Minneapolis.

The Effect of Alimentary Induced Hyperlipemia on Platelets. JACK W. COLE, ARCHIE G. BRITT, and WILLIAM D. HOLDEN, Cleveland.

An Experimental Study of the Proposed Mechanisms of Mast Cell Regulation in Relation to Blood Coagulation Theory. J. E. DEVITT and P. B. SAMUELS, Montreal.

Neurosurgery

Thursday, 9:00 a.m.—12:00 noon, South Ball Room, Conrad Hilton Hotel

Localization of Brain Tumors and Other Intracranial Lesions with Radioactive Iodinated Human Serum Albumen. HOWARD S. DUNBAR and BRONSON S. RAY, New York.

A Method of Measuring the Blood Flow of the Cerebral Cortex and the Effect of Carotid Ligation. JOHN E. ADAMS, DANIEL MEUB and KENNETH SCOTT, San Francisco.

Investigations of Physiologic Effects of Resection of Large Areas of the Brain in Humans. L. A. FRENCH and D. R. JOHNSON, Minneapolis.

A Study of Diaphragmatic Respiration in the Quadriplegic Patient. G. S. CAMERON, J. W. SCOTT, A. T. JOUSSE, and E. H. BOTTERELL, Toronto.

Myelography by Means of Radioiodine. ERIC T. YUHL and FRANZ K. BAUER, Los Angeles.

Effect of Intrathecal Radioactive Colloidal Gold. FREDERICK W. L. KERR and WILLIAM B. SEAMAN, St. Louis.

The Use of Trypsin Preparations for the Prevention and Lysis of Adhesions in the Subarachnoid Space. EDGAR A. BERING, JR., ROBERT L. MCLAURIN, PETER H. SCHURR, and FRANC D. INGRAHAM, Boston.

Quantitative Determination of Acceleration and Intracranial Pressure Produced at Impact in Experimental Head Injury. B. F. HADDAD, FREDERICK R. LATIMER, JOHN E. WEBSTER, H. R. LISSNER, and E. S. GURDJIAN, Detroit.

The Use of Parasympatholytic Drugs in the Treatment of Craniocerebral Injuries. DANIEL RUGE, Chicago.

Electrical Activity in Nerve and Thalamus Following Experimental Nerve Injury. WILLIAM E. HUNT and HENRY G. SCHWARTZ, St. Louis.

The Effect of Splanchnicectomy on the Urinary Output of Sodium by Hypertensive Patients. JESSE E. THOMPSON and REGINALD H. SMITHWICK, Boston.

Form Fitting Plastic Cranioplasty. WILLIAM T. SPENCE, Washington.

Peripheral Circulation and Shock

Thursday, 9:00 a.m.–12:00 noon, 8th Street Theatre

Observations on the Venous Circulation Time in the Lower Extremities. OSCAR CREECH, JR., PAUL F. PAULSON, JR., FRANK J. KELLY, and MICHAEL E. DEBAKEY, Houston.

Effect of Slow and Rapid Thawing on Prolonged Freezing and Chilling of Dog's Extremities. MARTIN A. ENTIN, Montreal.

Radiographic Observations on the Arterial and Venous Flow in Experimental Local Cold Injury. W. J. PIROZYNSKI and DONALD R. WEBSTER, Montreal.

A Clinical Method of Predicting the Healing of Amputations of the Ischemic Foot. FRANK H. LEEDS, RUTHERFORD S. GILFILLAN, NORMAN E. FREEMAN, and HELIO M. COELHO, San Francisco.

Is the Stress-Induced Inhibition of Wound Healing Due to an Excessive Endogenous Secretion of Adrenal Glands? JAMESON L. CHASSIN, H. A. MCDOUGALL, M. MACKAY, and S. ARTHUR LOCALIO, New York.

Wound Bleeding During Experimental Transfusion Reactions in Isoimmunized Dogs. STANLEY R. FRIESEN, CHARLES H. MCCROSKEY, and MARGARET ROADY, Kansas City, Kansas.

Experimental Citrate Intoxication During Massive Blood Transfusions. E. WATKINS, Boston.

Intraluminal Aortic Occlusion as a Possible Mechanism of Controlling Massive Intra-abdominal Hemorrhage. W. STERLING EDWARDS and PAUL P. SALTER, JR., Birmingham, Alabama.

Coronary Blood Flow and Myocardial Metabolism in Shock. W. STERLING EDWARDS, WILLY E. REBER, and RICHARD J. BING, Birmingham, Alabama.

Studies in Therapy of Hemorrhagic Shock with Associated Myocardial Damage. L. G. HAMPSON, H. J. SCOTT, and FRASER N. GURD, Montreal.

Internal Rates of Water Exchange in Patients Suffering from Shock Due to Hemorrhage, Salt Water Deficit, Burns, and Other Forms of Trauma. MORRIS J. FOGELMAN and BEN J. WILSON, Dallas.
Blood Volume Changes Following Battle Injuries. JOHN M. OLNEY, JR., THEODORE C. PRENTICE, WILLIAM H. CROSBY, and JOHN HOWARD, Washington.
A Comparison of the Metabolic Response to Soft Tissue and Bone Injury. ALLAN MYRDEN, GRAHAM WILSON, and FRANCIS D. MOORE, Boston.
Fenestration of Abdominal Wall; Its Application for Observation of Fibroplastic Effect of Plastics. JOSEPH K. NARAT, JOSEPH P. CANGELOSI, and JOHN V. BELMONTE, Chicago.

SYMPOSIUM ON TRAUMA

Tuesday, 2:00–5:00 p.m., Eighth Street Theatre

R. ARNOLD GRISWOLD, M.D., F.A.C.S., Louisville, Chairman, Committee on Trauma, American College of Surgeons, *Presiding.*
Peripheral Nerve Injuries. BARNES WOODHALL, M.D., F.A.C.S., Durham.
Amputations. RUFUS H. ALLDREDGE, M.D., F.A.C.S., New Orleans.
Fractures of the Ankle. GEORGE CURRY, M.D., F.A.C.S., Flint.
Rehabilitation. ALEXANDER P. AITKEN, M.D., F.A.C.S., Boston.
Trauma in a Rural Area. HOWARD E. SNYDER, M.D., F.A.C.S., Winfield, Kansas.
Plasma Expanders. GLEN J. COLLINS, COLONEL, M.D., Washington, D.C.

SYMPOSIUM ON CANCER

Wednesday, 2:00–5:00 p.m., John B. Murphy Memorial Auditorium

EDWIN P. LEHMAN, M.D., F.A.C.S., Charlottesville, Chairman, Committee on Cancer, American College of Surgeons, *Presiding.*
Review of the Chemotherapy of Cancer. DANELY P. SLAUGHTER, M.D., F.A.C.S., Chicago.
Cancer of the Esophagus. RICHARD H. SWEET, M.D., F.A.C.S., Boston.
Second Look. OWEN H. WANGENSTEEN, M.D., F.A.C.S., Minneapolis.
Bladder Cancer. VICTOR R. MARSHALL, M.D., F.A.C.S., New York.
Breast Tumors. NORMAN TREVES, M.D., F.A.C.S., New York.

SYMPOSIUM ON GRADUATE TRAINING IN SURGERY AND THE SURGICAL SPECIALTIES

Thursday, 1:30–3:00 p.m., North Ballroom, Conrad Hilton

MOTION PICTURE SYMPOSIUM ON UNUSUAL CONDITIONS ENCOUNTERED IN ABDOMINAL SURGERY

Thursday, 8:00–10:00 p.m., Grand Ballroom, Conrad Hilton

ANNUAL MEETING—BOARD OF GOVERNORS

Luncheon, Sunday, 10:00 a.m.–5:00 p.m., North Ballroom, Conrad Hilton Hotel, Chicago

WILLARD H. PARSONS, M.D., F.A.C.S., Vicksburg, Mississippi; Chairman, Board of Governors, *Presiding.*
Minutes of 1952 Annual Meeting of the Board of Governors. R. KENNEDY GILCHRIST, M.D., F.A.C.S., Chicago; Secretary, Board of Governors.
Report of the Chairman—Résumé of Interim Activities. WILLARD H. PARSONS, M.D., F.A.C.S.
Report of the Executive Committee. R. KENNEDY GILCHRIST, M.D., F.A.C.S., Secretary.
Report of the President. HAROLD L. FOSS, M.D., F.A.C.S., Danville, Pennsylvania.
Report of the Chairman of the Board of Regents. EVARTS A. GRAHAM, M.D., F.A.C.S., St. Louis.
Report of The Director. PAUL R. HAWLEY, M.D., F.A.C.S. (Hon.), Chicago.

Report of Committee to Study Revision of Bylaws as Suggested by the Governors. LELAND S. MCKITTRICK, M.D., F.A.C.S., Brookline; Chairman.
Report of Committee to Study the Method of Election of Governors. JAMES E. M. THOMSON, M.D., F.A.C.S., Lincoln, Nebraska; Chairman.
Report of the Committee on Malpractice Insurance. DONALD G. TOLLEFSON, M.D., F.A.C.S., Los Angeles; Chairman.
Report of Committee Concerning Increasing the Interest in the College of the Various Subspecialty Groups. CHARLES C. HIGGINS, M.D., F.A.C.S., Cleveland; Chairman.
Resolutions Presented by Individual Governors.
Announcement—Dinner that Evening and Adjourned Meetings of the Board of Governors.

ADJOURNED MEETING—BOARD OF GOVERNORS

Thursday, 3:30–3:45 p.m., Grand Ballroom, Conrad Hilton Hotel

WILLARD H. PARSONS, M.D., F.A.C.S., Chairman; *Presiding.*
Report of Committee on Nominations to the Board of Governors.
Report of Nominating Committee on Election of Regents.
Election of Regents of College.
Report of Nominating Committee on Election of Officers of Board of Governors.
Election of Officers of Board of Governors.
Announcement of Nominating Committee, Board of Governors 1953–1954.
Adjournment.

ANNUAL MEETING OF FELLOWS

Thursday, 3:45–6:00 p.m., Grand Ballroom, Conrad Hilton

HAROLD L. FOSS, M.D., F.A.C.S., Danville, Pa.; President, *Presiding.*
Report of Committee on Nominations:
Election of Officers.
Election of Governors.
Deliberations, 1953 Annual Meeting, Board of Governors. WILLARD H. PARSONS, M.D., F.A.C.S., Vicksburg, Mississippi, Chairman, Board of Governors.
Report of the Treasurer. WARREN H. COLE, M.D., F.A.C.S., Chicago, Treasurer.
Committee on Cancer. EDWIN P. LEHMAN, M.D., F.A.C.S., Charlottesville, Chairman.
Committee on Trauma. R. ARNOLD GRISWOLD, M.D., F.A.C.S., Louisville, Chairman.
Committee on Graduate Training in Surgery. FREDERICK A. COLLER, M.D., F.A.C.S., Ann Arbor, Chairman.
Committee on Medical Motion Pictures. HILGER PERRY JENKINS, M.D., F.A.C.S., Chicago, Chairman.
Committee on Nutrition of Surgical Patient. WALTER G. MADDOCK, M.D., F.A.C.S., Chicago, Chairman.
American College of Surgeons, Summary of Annual Departmental Reports. PAUL R. HAWLEY, The Director.
General Discussion.
Adjournment.

FUTURE CLINICAL CONGRESSES

1954	Atlantic City, New Jersey	October 25 to 29
1955	Chicago, Illinois	October 31 to November 4
1956	San Francisco	October 8 to 12

THE CLINICAL CONGRESS TECHNICAL EXHIBITION

The Technical Exhibition, to be located at the Conrad Hilton Hotel, Chicago, provides an attractive method of displaying supplies and equipment such as surgical instruments, diagnostic and therapeutic apparatus, pharmaceuticals, medical literature, and other similar items designed for use in hospitals and surgeons' offices. The list of exhibitors follows:

Abbott Laboratories, North Chicago
Air-Shields, Inc., Hatboro, Pa.
W. D. Allison Company, Indianapolis, Ind.
A. S. Aloe Company, St. Louis, Mo.
American Cystoscope Makers, Inc., New York
American Hospital Supply Corporation, Evanston, Ill.
American Safety Razor Corporation, Brooklyn
American Sterilizer Company, Erie, Pa.
Ames Company, Inc., Elkhart, Ind.
Appleton-Century-Crofts, Inc., New York
The Armour Laboratories, Chicago
Arnar-Stone Laboratories, Inc., Evanston, Ill.
Association of American University Presses, New York
Austenal Laboratories, Inc., New York
Ayerst, McKenna & Harrison Limited, New York
C. R. Bard, Inc., Summit, N. J.
Bard-Parker Company, Inc., Danbury, Conn.
Bauer & Black, Chicago
W. A. Baum Co., Inc., Copiague, L. I., N. Y.
Rudolph Beaver, Inc., Waltham, Mass.
Becton, Dickinson and Company, Rutherford, N. J.
Berivon Company, Meridian, Miss.
The Burdick Corporation, Milton, Wis.
Cameron Surgical Specialty Company, Chicago
S. H. Camp and Company, Jackson, Mich.
Campbell Associates, Cincinnati
Wilmot Castle Company, Rochester, N. Y.
Chesebrough Manufacturing Co., Cons'd., New York
Gilbert Hyde Chick Company, Oakland, Calif.
Chilean Iodine Educational Bureau, Inc., New York
Ciba Pharmaceutical Products, Inc., Summit, N. J.
Clay-Adams Company, Inc., New York
The Coca-Cola Company, Atlanta
Cochrane Physicians' Supplies, Inc., New York
Codman & Shurtleff, Inc., Boston
C. S. C. Pharmaceuticals, New York
Continental Medical Bureau, Los Angeles
Coreco Research Corporation, New York
Cutter Laboratories, Berkeley, Calif.
F. A. Davis Company, Philadelphia
Davis & Geck, Inc., Brooklyn
Davol Rubber Company, Providence, R. I.
J. A. Deknatel & Son, Inc., Queens Village, L. I., N. Y.
De Puy Manufacturing Co., Inc., Warsaw, Ind.
Desitin Chemical Company, Providence, R. I.
Duke Laboratories, Inc., Stamford, Conn.
Eaton Laboratories, Inc., Norwich, N. Y.
Eisele & Company, Nashville
Electro Therapeutic Instrument Company, Chicago
Ethicon Suture Laboratories, Inc., New Brunswick
Harold L. Feighner & Company, Detroit
Fenwal Laboratories, Inc., Framingham, Mass.
Charles O. Finley Company, Chicago
C. B. Fleet Co., Inc., Lynchburg, Va.
Florida Brace Corporation, Winter Park, Fla.
E. Fougera & Company, Inc., New York
Geigy Pharmaceuticals, New York
Gomco Surgical Manufacturing Corp., Buffalo
John F. Greer Company, Oakland, Calif.
Grieshaber Manufacturing Company, Chicago
Grune & Stratton, Inc., New York
Gudebrod Bros. Silk Co., Inc., New York
Hanger Artificial Limbs, Washington, D. C.
Paul B. Hoeber, Inc., New York
Hoffmann-La Roche, Inc., Nutley, N. J.
Huntington Laboratories, Inc., Huntington, Ind.
Identical Form, Inc., New York
Jobst Applied Biomechanics Institute, Toledo
Johnson & Johnson, New Brunswick
Kansas City Assemblage Company, Kansas City, Mo.
La Resista Corset Company, Bridgeport, Conn.
Lea & Febiger, Philadelphia
Lederle Laboratories Division, New York
Eli Lilly and Company, Indianapolis
J. B. Lippincott Company, Philadelphia
P. Lorillard Co., Inc., New York
The Macmillan Company, New York
T. J. McGuire Travel Company, Chicago
Mead Johnson & Company, Evansville, Ind.
Medco Products Company, Tulsa
Medical Aids, Inc., Chicago
The Medical Bureau, Chicago
Medical Case History Bureau, New York
Merck & Co., Inc., Rahway, N. J.
Miles Laboratories, Inc., Elkhart, Ind.
Morgan's, Incorporated, Chicago
C. V. Mosby Company, St. Louis
V. Mueller & Company, Chicago
National Electric Instrument Co., Inc., Elmhurst, N. Y.
Novocol Chemical Mfg. Co., Inc., Brooklyn
O-Cel-O, Buffalo
Ohio Chemical & Surgical Equipment Co., Madison, Wis.
Orthopedic Equipment Company, Bourbon, Indiana
Orthopedic Frame Company, Kalamazoo, Mich.
Ortho Pharmaceutical Corporation, Raritan, N. J.
Parke, Davis & Company, Detroit
Chas. Pfizer & Co., Inc., Brooklyn
Pharmaseal Laboratories, Glendale, Calif.
George P. Pilling & Son Company, Philadelphia
Richards Manufacturing Co., Memphis
Ritter Company, Inc., Rochester, N. Y.
Robbins Instrument Corporation, Attleboro, Mass.
Rystan Company, Inc., Mount Vernon, N. Y.
W. B. Saunders Company, Philadelphia
G. D. Searle & Co., Chicago
Shampaine Company, St. Louis
Sharp & Dohme, Philadelphia
Siebrandt Fracture Equipment, Inc., Kansas City, Mo.
J. Sklar Manufacturing Co., Long Island City, N. Y.
Smith, Kline & French Laboratories, Philadelphia.
Spencer, Incorporated, New Haven
E. R. Squibb & Sons, Long Island City, N. Y.
The Stuart Company, Chicago
Surgery, Gynecology and Obstetrics, Chicago
Taylor Instrument Companies, Rochester, N. Y.
The Tower Company, Inc., Seattle
United Surgical Supplies Company, Mamaroneck, N. Y.
Vestal, Incorporated, St. Louis
Wallace & Teirnan Company, Inc., Newark
Edward Weck & Company, Inc., Brooklyn
The Williams & Wilkins Company, Baltimore
Winthrop-Stearns, Inc., New York
Woodward Medical Personnel Bureau, Chicago
Wyeth Laboratories, Philadelphia
The Year Book Publishers, Inc., Chicago
Zimmer Manufacturing Company, Warsaw, Ind.